*MEASUREMENT AND PROGNOSTIC SIGNIFICANCE OF POSTERIOR URETHRA TO BULBAR URETHRAL RATIO IN BOYS WITH NEUROGENIC BLADDER ….*and its comparison with the ratio in posterior urethral valves and male controls.

A dissertation submitted in partial fulfillment of M.Ch branch V (Paediatric Surgery) examination of The Tamilnadu Dr.M.G.R Medical University,Chennai, to be held in August 2011.

CERTIFICATE

This is to certify that the dissertation entitled **"Measurement and prognostic significance of Posterior urethra to bulbar urethral ratio in boys with neurogenic bladder and its comparison with the ratio in posterior urethral valves and male controls"** submitted in partial fulfillment of the regulation for the **MCh Branch-V (Paediatric Surgery)**, examination of the Tamilnadu Dr MGR Medical University,Chennai to be held in August 2011, is the bonafide original work of **Dr Kshama Vasudev Kulkarni**, Senior Postgraduate student in the Department of Paediatric surgery,Christian Medical College,Vellore under my guidance and supervision .This dissertation has not been submitted ,fully or in part to any other board or university.

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INTRODUCTION

Micturating cystourethrogram is a basic investigation in three important urological conditions in children - 1.Posterior urethral valves (PUV) 2.Neurogenic bladder (NB) and 3.Vesicoureteric reflux (VUR). While on the one hand these conditions may mimic one another, on the other, there is a degree of true overlap as in reflux secondary to dysfunctional voiding, neurogenic bladder and posterior urethral valves. Therefore it is imperative to make a correct diagnosis in these cases and to differentiate these conditions from each other as the management of each condition is different.

Ureteric reimplantation is the primary surgical treatment in vesicoureteric reflux, but is of secondary importance in neurogenic bladder and posterior urethral valves. Similarly, fulguration is the treatment of choice in posterior urethral valves, but is harmful in neurogenic bladder, misdiagnosed as posterior urethral valves. It is not uncommon to misdiagnose neurogenic bladder as PUV because a dilated urethra is seen in both and in the absence of clinically recognizable neural stigmata like meningomyelocoele (MMC) and sacral agenesis, these cases can be misdiagnosed as PUV and treated inappropriately. This is especially important when we are dealing with nonneurogenic neurogenic bladder (NNNB) cases.

While the posterior urethral dilatation in PUV has been quantified in the recent literature [1, 2, 3] similar quantification is not available in neurogenic bladder. This study is an attempt to quantify the posterior urethral dilatation on micturating

cystourethrograms in boys with neurogenic bladder and compare it with that in posterior urethral valves and in boys who have had a micturating cystourethrogram but neither had neurogenic bladder nor posterior urethral valves (control group).

AIMS AND OBJECTIVES

1. To assess the posterior urethral dilatation on micturating cystourethrogram by using posterior urethra to bulbar urethral ratio in boys with neurogenic bladder and to compare it with that in posterior urethral valves and normal controls.

2. To assess whether there is any correlation between upper tract changes in neurogenic bladder patients and the dilatation of posterior urethra, as quantified by using urethral ratio.

3. To assess whether the urethral ratio in neurogenic bladder patients has any correlation with the age of the child at first diagnosis.

4. To assess whether posterior urethral dilatation is any different in neurogenic bladder caused by meningomyelocoele verses sacral agenesis.

5. To assess whether posterior urethral dilatation has any association with bladder compliance in neurogenic bladder.

REVIEW OF LITERATURE

EMBRYOLOGY OF MALE URETHRA:

The embryology of urethra is not completely understood, particularly as it pertains to pathologic anomalies. The proximal or posterior urethra is formed by differentiation of the urogenital sinus, and the anterior urethra results from tubularization of the urethral plate.

The urethra is usually divided into four sections:

1. The prostatic urethra, which is defined from the bladder neck to the proximal portion of urogenital diaphragm.
2. The membranous urethra or that portion that traverses the diaphragm
3. The bulbar urethra that segment from the membranous urethra to the penoscrotal junction
4. The penile urethra which traverses the penile shaft and the glans.

Between the fourth and seventh week of gestation the cloaca subdivides into a posterior portion (anorectal canal) and an anterior portion (primitive urogenital sinus).The urogenital sinus then develops into a cranial portion that dilates to form the urinary bladder and pelvic portion that forms the proximal prostatic urethra and the membranous urethra .The anterior urethra is formed from the urethral folds on the genital tubercle. That portion of the urethra unlike the posterior urethra is dependent on 5 alpha reductase [4].

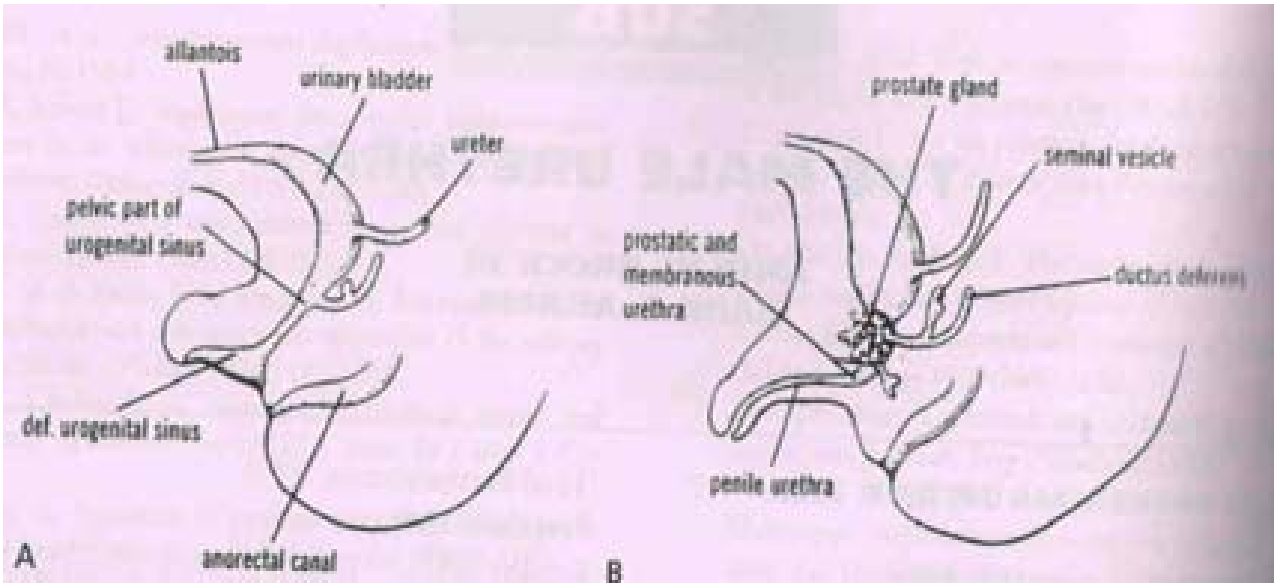


Fig 1: Development of male urethra

STRUCTURAL ANATOMY OF LOWER URINARY TRACT:

The whole anatomic unit that stores and eliminates urine is called bladder and the smooth muscle in the bladder wall is called the detrusor [5]. The detrusor consists of numerous interlacing muscle bundles that interdigitate with one another in an intricate fashion, resulting in a complex meshwork of smooth muscle [6]. There are no identifiable continuously separate layers of the detrusor. This arrangement is such that when the detrusor contracts there is reduction in the size of the bladder in all dimensions, resulting in efficient emptying. The muscle fibers of the detrusor continue into the bladder neck and also surround the proximal urethra. Although the muscles of the detrusor and bladder neck are smooth, unlike other types of smooth muscle they are under voluntary control.

The urethral sphincter mechanism is composed of smooth and striated muscle components. The smooth muscle component consists of the continuation of the detrusor

into the vesical neck and proximal urethra. This is called the internal urethral sphincter. The striated component surrounds and is integral with the urethra where it passes through the urogenital diaphragm. This is called the external urethral sphincter and is composed of both slow and fast twitch muscle fibers .The slow twitch capability of these fibers allows a more sustained contraction than normal striated muscle and the striated sphincter is therefore able not only to close the urethra acutely but also to maintain passive continence for prolonged periods of time.

Because the smooth (internal) and striated (external) sphincters have different nerve supplies, their function in providing urinary continence is independent of each other. The smooth muscle sphincter at the bladder neck (internal sphincter) is however the primary continence mechanism [7].

In a normal bladder intravesical pressure during voiding is exerted evenly against all points of the internal bladder wall. This pressure compresses the submucosal portion of the distal ureter against the detrusor behind it resulting in a functional closure that prevents reflux.

The normal bladder has viscoelastic properties that allow it to stretch significantly without an attendant rise in tension. Positional changes of the patient or increased abdominal pressure occurring with straining or coughing result in a significant rise in the intravesical pressure; however because the increased pressure is distributed equally to both bladder and the urethra, there is no net change in the pressure gradient between the two, and continence is preserved [5].

NEUROANATOMY OF NORMAL VOIDING:

The micturition reflex is a complicated neurologic event governing voiding that requires integration of multiple neural pathways.

Pontine micturition center:

Integration of the micturition reflex occurs in the pontine micturition center, located in the brain stem. A number of suprapontine pathways connect the cerebral cortex with the pontine micturition center, and provide voluntary cerebral control over the micturition reflex.

Sacral micturition center:

It is located in the second through fourth sacral cord segments, and a pathway in the spinal cord connects it with the pontine micturition center. Another neural pathway in the spinal cord connects the motor cortex of the brain with the pudendal nucleus, which also is located in the second through fourth sacral cord segments; the purpose of this pathway is to enable voluntary contraction of the external sphincter by the pudendal nerve [5].

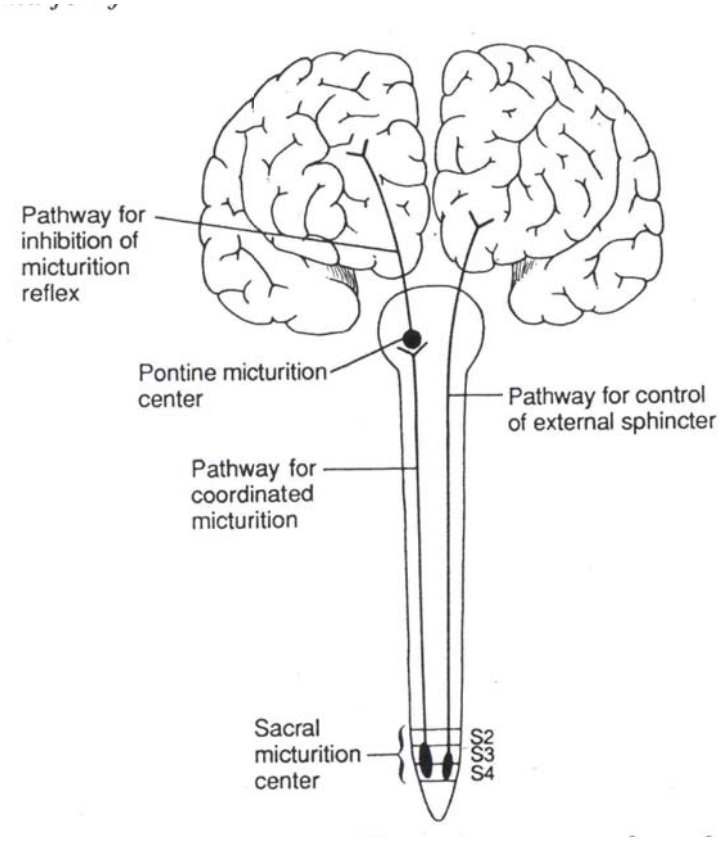


Fig 2: Neuroanatomy of brain, brain stem, and spinal cord neural pathways involved micturition.

Peripheral nerves:

Activation, coordination, and integration of various parts of the bladder-sphincteric complex involves both the central somatic and autonomic nervous systems through three sets of peripheral nerves: sacral parasympathetic (pelvic nerve), thoracolumbar sympathetic (hypogastric nerves and sympathetic chain), and sacral somatic nerves (primarily the pudendal nerve) [8-9].

Parasympathetic nerve fibers run in the pelvic nerve (S2 to S4) to supply the pelvic and vesical plexuses before entering the bladder. Parasympathetic ganglia are found within these plexuses and in the bladder wall. Sympathetic nerves arise from

segments T10 to L2 of the spinal cord and go to the inferior mesenteric ganglion through the sympathetic trunk. From the inferior mesenteric ganglion the nerve fibers pass to the pelvic plexus and bladder through the hypogastric nerves. There is also sympathetic innervation originating from T10 to L2 supplying the detrusor and urethral sphincter [10]. The somatic nervous system (pudendal nerve) supplies the periurethral pelvic floor musculature [9]. The sensory and motor nerves carried by all three nerves innervate both the bladder and urethral sphincter. They originate from parasympathetic ganglia located in the second, third, and fourth segments of the sacral spinal cord [10]. Within the spinal cord, information from bladder afferents is integrated with that from other viscera and somatic sources and projected to the brain stem centers that coordinate the micturition cycle [11].

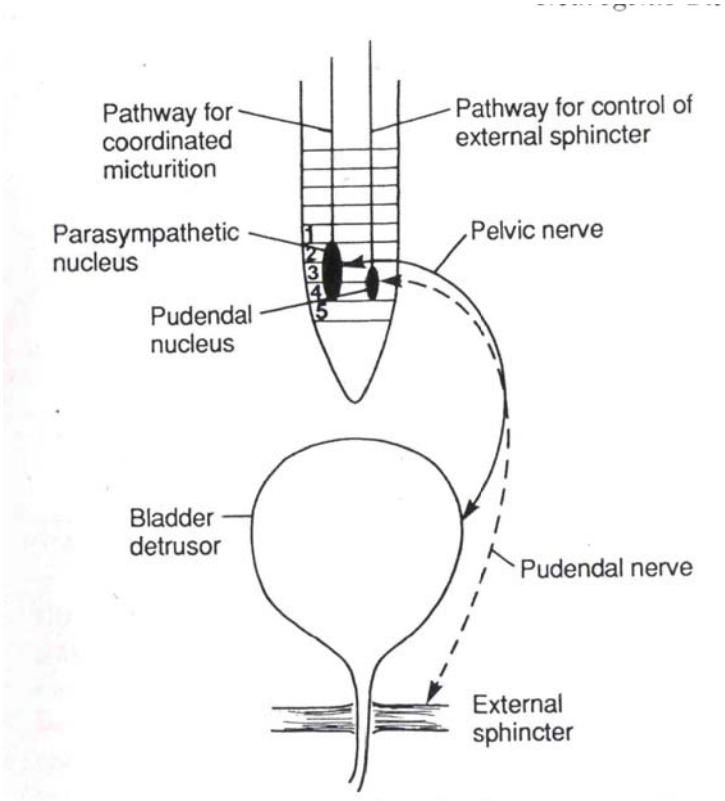


Fig 3: Neuroanatomy of sacral reflex arc.

Although detrusor control is predominantly parasympathetic, the sympathetic nervous system can block parasympathetic conduction, preventing premature detrusor contractions and maintaining tone of the internal sphincter at the bladder neck. The sympathetics therefore, serve to modulate the storage of urine [5].

NEUROPHYSIOLOGY OF NORMAL VOIDING:

Normally as the bladder fills there is a complex, unconscious inhibition of the micturition reflex by the suprapontine pathway. As the bladder distends to near its capacity, a need to void is transmitted to sensory centers in the brain. Then, when voiding is necessary and can occur in a socially acceptable situation, the micturition reflex is consciously activated.

The micturition reflex allows coordinated voiding, which involves, in the following order, relaxation of the external urethral sphincter, contraction of detrusor and finally opening of the internal sphincter. During voiding sympathetic pathways are deactivated; also, opening of the internal sphincter results in funneling of the bladder base. Pressure in the widely patent urethra during voiding is the same as that in the bladder. Interruption of the urinary stream may be accomplished by voluntary contraction of the external urethral sphincter. Any urine remaining in the proximal urethra is then milked back into the bladder by the internal sphincter [5].

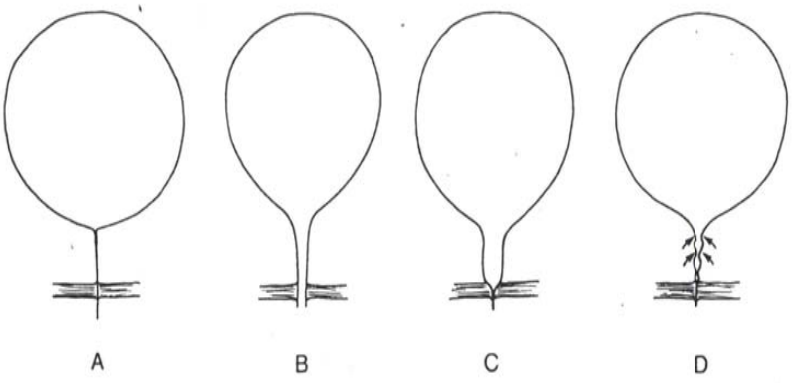


Fig 4: Diagram of normal voiding. A - In the normal continent state the urethra is closed throughout by sphincters at the bladder neck and urogenital diaphragm. B - During normal voluntary voiding, the external sphicters at the level of the urogenital diaphragm relaxes, the bladder detrusor muscle contracts,and the bladder neck funnels.Pressure during voiding is the same in the bladder lumen and urethra. C - Upon completion of voiding, the external sphincter is consciously contracted. D - Urine remaining in the proximal urethra is milked back into the bladder by the internal sphincter fibers that surround this area.

Under normal conditions, the detrusor muscle, bladder neck, and striated external sphincter function as a synergistic unit for adequate storage and complete evacuation of urine. When a neurourologic lesion exists, these components usually fail to act in unison.

Multiple classification systems have been proposed to link these various neurologic disease processes and to provide and their resultant voiding dysfunction [12]. The classification based on the reflexivity of the detrusor is a gross simplification of neurogenic bladder and allows understanding the basic pathophysiology involved in lesions at various levels. Simply put, lesions of the sacral reflex arc (lower motor neuron)

typically result in detrusor areflexia; lesions above this level result in detrusor hyperreflexia [5].

1. Detrusor Areflexia:

Typically, it is secondary to neurologic lesions affecting the sacral micturition center or pathways connecting this center with the bladder (or both). In most cases the urethral sphincter mechanism remains competent. Because there is no perception of bladder distension, the bladder will fill until the viscoelastic properties of the smooth muscle fibers in its wall are exceeded; intravesical pressure then rises and eventually exceeds that exerted by the urethral sphincter mechanism. At this point overflow incontinence occurs until the pressures are equalized, at which point incontinence ceases.

In yet other cases of detrusor areflexia the sphincter mechanism may be compromised, resulting in a continual dribbling incontinence of urine. If some urethral sphincter activity remains, however, the patient can maintain continence by emptying the bladder regularly with abdominal straining or the credes maneuver. The amount of residual urine is dependent on the effectiveness of these adjunctive voiding mechanisms.

Conditions that result in detrusor areflexia include lower spinal cord tumors, herniated intervertebral disc, trauma or pelvis surgery that damage the pelvic or pudendal nerves (or both) of the sacral reflex arc.

2. Detrusor Hyperreflexia:

There are two subgroups of detrusor hyperreflexia.

a) **Lesion in cerebral cortex**: Causes uninhibited bladder contractions, but micturition reflex is intact, and voiding, even when completely involuntary, is physiologically coordinated.

b) **Lesion in suprasacral spinal cord**: Micturition reflex is disrupted and voiding, when it occurs, is frequently uncoordinated, because of Detrusor external sphincter dyssynergia (DESD)

Detrusor hyperreflexia caused by lesions of the suprasacral spinal cord are more ominous because of the potential for upper urinary tract damage. DESD occurs in up to three fourths of patients with suprasacral spinal cord lesions.

In DESD the external urethral sphincter contracts involuntarily at the same time a bladder detrusor contraction occurs, thus impeding urinary flow because of increased urethral resistance [13]. Normal contractions of the external urethral sphincter are voluntary and allow purposeful interruption of the urinary stream. With disruption of the suprasacral pathways, the normal coordination between the detrusor and the external sphincter often is not possible. When this type of uncoordinated voiding occurs, intravesical pressure may become very high. This increased pressure is a major factor in causing upper urinary tract deterioration.

Common neurologic disorders of the suprascaral spinal cord resulting in this type of detrusor hyperreflexia include dysraphic myelodysplasia, spinal cord trauma and spinal cord tumors.

PATHOPHYSIOLOGY OF UPPER TRACT CHANGES IN NEUROGENIC BLADDER:

In patients with NBSD, disordered innervation of the detrusor musculature and external sphincter adversely affects bladder function. Children with this condition can be categorized into high-risk and low-risk groups for secondary damage from a neurogenic bladder based on intravesical pressure. When the detrusor (filling) pressure exceeds 40 cm H_2O, glomerular filtration rate decreases and pyelocaliceal and ureteral drainage deteriorates, leading to obstructive hydronephrosis and/or vesicoureteral reflux [14-17]. Even in the absence of reflux or upper urinary tract dilatation, high intravesical pressure can impair drainage of urine into the bladder. Any pathophysiologic process that causes either intermittent or continuous elevation of bladder pressure above 40 cm H_2O places the child at risk for upper urinary tract dysfunction, urinary tract infections, and ultimately renal failure. Intermittent elevation of bladder pressure may occur from detrusor hypertonia, hyperreflexia, or both. Hyperreflexia may cause intermittent elevation of bladder pressure, especially if the external sphincter acts reflexively and tightens rather than relaxes in an attempt to prevent micturition [detrusor external sphincter dyssynergia (DESD)]. Over a long period of time, hyperreflexia with pressures greater than 40 cm H_2O may result in detrusor decompensation (areflexia from myogenic failure) or in detrusor hypertrophy with associated sacculations and subsequent diverticula formation. These pathophysiologic changes affect the elastic and vesicoelastic properties of the bladder and also result in mechanical ureterovesical junction obstruction. Continuous elevation of bladder pressure above 40 cm H_2O may occur from

a hypertonic detrusor or a hypertrophic small-capacity bladder secondary to outflow obstruction [18]. Bladder outlet obstruction is caused by DESD, or by fibrosis of the external urethral sphincter secondary to partial or complete denervation [19-21]. Bladder outlet obstruction will lead to elevated (pathologic) voiding pressures, which will contribute to either detrusor decompensation or hypertrophy. Finally, recurrent urinary tract infections due to bladder residue may aggravate damage to the neurogenic bladder through processes of transmural inflammation and fibrosis. Together with high intravesical pressures and/or vesicoureteral reflux, these lower urinary tract infections will lead to episodes of acute pyelonephritis and irreversible renal damage.

PATHOPHYSIOLOGY OF URETHRAL DILATION IN NEUROGENIC BLADDER:

Posterior urethral dilation is mainly seen in patients with neurogenic bladder who have associated detrusor external sphincter dyssynergia (DESD).In these patients, during voiding, the external sphincter at the level of urogenital diaphragm contracts involuntarily and inappropriately. This lack of co-ordination between the bladder detrusor and external sphincter contractions causes functional outlet obstruction at the level of membranous urethra [5], causing increased pressure in the proximal urethra (as well as in the bladder lumen), that results in significant widening of the prostatic urethra and resultant dilatation [4].

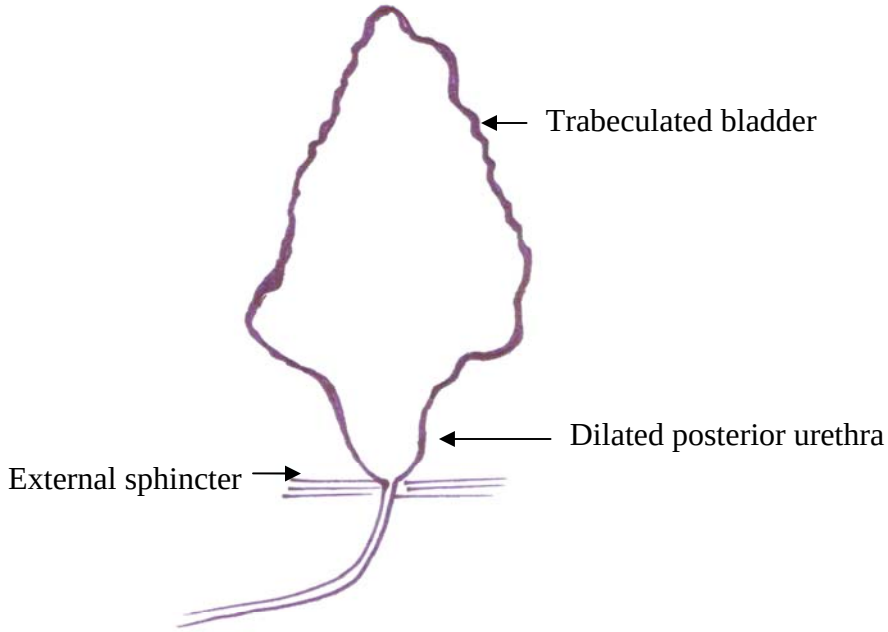


Fig 5: Neurogenic bladder with detrusor external sphincter dyssynergia.

The posterior urethral dilatation can be seen in other conditions affecting the vesicourethral unit, as in posterior urethral valves and in nonneurogenic neurogenic bladder. Though the pathophysiology of posterior urethral dilatation in these conditions may be different, on imaging studies they may simulate each other, and hence can cause diagnostic confusion.

PATHOPHYSIOLOGY OF URETHRAL DILATION IN PUV:

In patients with posterior urethral valves, there is mechanical obstruction to the passage of urine due to presence of valves in the prostatic urethra. This leads high voiding pressures which distend and thin the prostatic urethra. The storage capacity of the prostatic urethra sometimes exceeds that of the bladder because of the relative lack of muscle there. The verumontanum is distorted, and the ejaculatory ducts may be dilated from refluxing urine. The bladder neck is rigid and hypertrophied. Bladder neck appearance and function usually improve after the obstructive valves are destroyed.

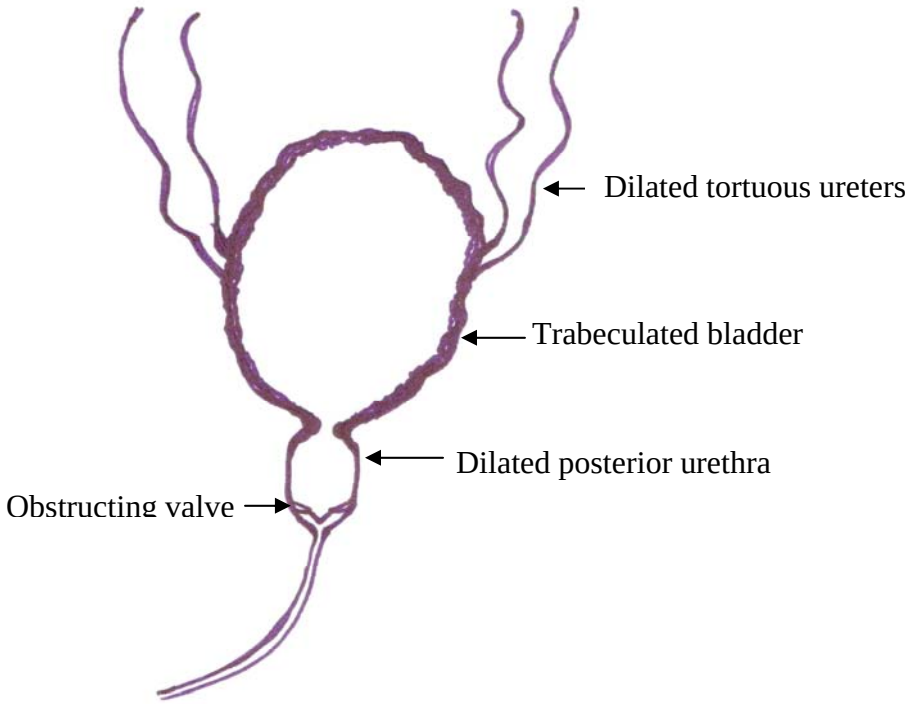


Fig 6: Posterior urethral valves

MICTURATING CYSTOURETHROGRAM FEATURES IN DIFFERENT CONDITIONS:

MCU in neurogenic bladder:

In children with neurogenic bladder dysfunction the easily recognized radiological findings on voiding cystourethrogram are most often attributable to dyssynergia between the detrusor muscle and external sphincter. They represent a combination of transient functional changes, that is irregular mucosal pattern, hourglass bladder configuration and saccular dilatation of the posterior urethra, as well as chronic morphological abnormalities, including contracted bladder, “pine-tree” shaped bladder ,trabeculation and pseudodiverticulum of the bladder wall [22]. With the recently developed understanding of neurogenic bladder and its treatment, in most cases the radiologic patterns of fully developed disease are no longer seen.

MCU in Nonneurogenic neurogenic bladder :

Hinman [23] described an apparent ‘syndrome’ of voiding dysfunction that mimics neuropathic bladder disease but may be a learned disorder. It is produced by an active contraction of the sphincter during voiding, creating a degree of outflow obstruction.

Radiologic features include large-capacity, trabeculated bladder,reflux: III/V in degree, large residual urine volume, posterior urethra sometimes dilated with narrowing at external sphincter.

MCU in Posterior urethral valves :

In PUV, the MCU often demonstrates bladder diverticula and severe vesicoureteral reflux. From the lateral projection, the bladder neck is elevated and the proximal urethra is dilated, and the actual valve structure is often visible.

Shopfner and Hutch as well as Popek et al have hypothesized that the posterior urethra has the capacity to stretch with increased voiding pressure due to its unique histological and anatomical configuration, in contrast to the anterior urethral segment [24, 25]. Hence, on micturating cystourethrogram, the posterior urethra may look dilated in neurogenic bladder, PUV and nonneurogenic neurogenic bladder, though the pathophysiology of posterior urethral dilatation is different in all these conditions. Therefore it is imperative to make a correct diagnosis in these cases and to differentiate these conditions from each other as the management of each condition is different.

Few authors have studied the changes in the anatomical configuration of posterior urethra pre and post fulguration in posterior urethral valves; however a comparison of the dilatation of posterior urethra in neurogenic bladder patients with that of posterior urethral valves and cystoscopically normal boys has not been studied so far.

O. Bani Hani et al from the Childrens Hospital at Westmead, Australia, performed a study on 23 infants to provide a ratio to measure successful treatment of posterior urethral valves. Median preoperative ratio was 8.6 and it decreased postoperatively to 3.1. They concluded that calculating urethral ratio in patients with posterior urethral valves

allows objective measurement of the technical success of valve ablation [1]. Similar study was carried out by Gupta et al in India, and they concluded that calculation of urethral ratio on VCUG for assessment of outcome of fulguration is objective, reproducible, and allows preoperative and postoperative VCUG from different facilities to be compared. In their study, mean urethral ratio in pre-fulguration group was 4.94 (SD 2.97) and they found that a post-fulguration urethral ratio of 2.5-3 represents an acceptable result postoperatively [2].

Menon et al from PGIMER Chandigarh, India, carried out a study on 217 patients to assess the morphological normalization of posterior urethra on MCUG 3 months after fulguration and correlated these changes with the overall clinical status of the patients. As the pre and post fulguration MCUG may be taken by different Xray machines and in different setup, the ratio of the diameter of PU to that of the BU was taken .This ratio is a useful tool in predicting severity of the disease and objectively assesses the adequacy of fulguration independent of the surgeon's opinion. They found a significant reduction in the dimensions of the posterior urethra and an increase in the dilatation of the anterior urethra, especially the bulbar urethra, after a successful valve fulguration. They concluded a post operative PU/BU ratio >3 SD(1.92) should alert to an incomplete fulguration or stricture and patients with normal range ratio have faster recovery of slow draining units ,reflux and less voiding dysfunction [3].

All these studies are done in PUV patient's pre and post fulguration, however so far no study has been published which has quantified the dilatation of posterior urethra in neurogenic bladder patients and compared it with that in PUV and cystoscopically normal urethra as in primary VUR.

URODYNAMICS IN NEUROGENIC BLADDER:

The applicability of urodynamic testing has expanded so much that most pediatric urologic centers consider functional assessment of the lower urinary tract an integral element in the evaluation process and as important as radiographic visualization in characterizing and managing these abnormal conditions [14, 19]. Urodynamic studies fulfill several objectives: [14, 19, 26, 27]

- They provide baseline information about the radiologic appearance of the upper and lower urinary tract, as well as the condition of the sacral spinal cord and the CNS.
- The studies can then be compared with later assessments, so that early signs of deteriorating urinary tract drainage and function, or of progressive neurologic denervation, can be detected.
- They help to identify babies at risk for urinary tract deterioration as a result of detrusor hypertonicity or outflow obstruction from detrusor–sphincter dyssynergy, which then allows prophylactic measures to be initiated before the changes actually take place.
- They help the physician to counsel parents with regard to their child's future bladder and sexual function.

Three categories of lower urinary tract dynamics:

1. Synergic.
2. Dyssynergic, with and without detrusor hypertonicity.
3. Complete denervation

1. ***Synergy*** is characterized by complete silencing of the sphincter during a detrusor contraction or when capacity is reached at the end of filling. Voiding pressures are usually within the normal range.
2. ***Dyssynergy*** occurs when the external sphincter fails to decrease, or actually increases its activity during a detrusor contraction or a sustained increase in intravesical pressure, as the bladder is filled to capacity [28]. Frequently, a poorly compliant bladder with high intravesical pressure is seen in conjunction with a dyssynergic sphincter, resulting in a bladder that empties only at high intravesical pressures [20].
3. ***Complete denervation*** is noted when no bioelectric potentials are detectable in the region of the external sphincter at any time during the micturition cycle or in response to a Credé maneuver or sacral reflex stimulation.

Categorizing lower urinary tract function in this way is extremely useful because it defines which children are at risk for urinary tract changes, who should be treated prophylactically, who needs close surveillance, and who can be followed at greater intervals without fear of deterioration.

On initial assessment or subsequent studies, 71% of newborns with dyssynergic voiding have urinary tract deterioration within the first 3 years of life, whereas only 17% of synergic voiders and 23% with completely denervated sphincter developed similar changes. Thus, it appears that outlet obstruction is a major contributor to the development of urinary tract deterioration in these children.

Although videourodynamics is the state-of-the-art modality for evaluating complex or refractory neurogenic bladder, it is currently available only in few centers in India, and hence many patients does not have access to this sophisticated study.

TREATMENT OF NEUROGENIC BLADDER:

At birth, the majority of patients with neurogenic bladder have normal upper urinary tracts. Without proper management, urinary tract infections and elevated bladder pressures with secondary bladder-wall changes may cause upper urinary tract deterioration within 3 years in up to 58% [29]. One third of children who develop impaired kidney drainage do so within the first year of life [30]. Crucial for long-term prognosis of patients with neurogenic bladder sphincter dysfunction is the fact that the management must start before consequences of bladder dysfunction become apparent.

The goals of management are to prevent or minimize secondary damage to the upper urinary tracts and bladder from the primary neurogenic bladder dysfunction and to achieve safe social continence [31]. Thus, long before continence becomes an issue, starting from the first year of life, management is directed at creating a low-pressure reservoir and ensuring complete and safe bladder emptying.

Clean intermittent catheterization (CIC) in combination with anticholinergics (oxybutynin) is the standard therapy for children with neurogenic bladder dysfunction with detrusor hyperactivity and/or DESD [18, 32, 33]. This treatment is also feasible and effective in developing countries, where untreated neuropathic bladder is an important

cause of preventable chronic renal failure [34, 35]. CIC enables complete bladder emptying and thus avoids bladder residues and consequent risks for infections. In the high-risk bladder with DESD, CIC also allows bladder emptying before the occurrence of otherwise “spontaneous” high-pressure voiding, which is known to be detrimental for kidney function and drainage. Parental acceptance of, and compliance with, clean intermittent catheterization appears to be far greater if it has been used consistently from birth.

Oxybutynin, a bladder smooth-muscle relaxant, is used to improve bladder dynamics through suppression of detrusor hypertonicity and hyperreflexia. By doing so, oxybutynin eliminates (high-pressure) uninhibited detrusor contractions (and thus urinary leakage) and prevents high-pressure bladder storage (due to detrusor hypertonicity or low bladder compliance) and high-pressure emptying (in case of DESD).

When a hyperreflexic or hypertonic bladder fails to respond to these measures, augmentation cystoplasty may be required. However, the need for this operative modality in children managed proactively has been substantially reduced to 17%, as compared with a 41% incidence in children followed expectantly [36,37]. Furthermore, the use of vesicostomy drainage has been almost completely eliminated since this approach has been adopted.

Given the success of other specialties (physical medicine and rehabilitation, orthopedics, and neurology) at improving and prolonging the lives of the neurologically

impaired patient,we as paediatric surgeons have an increasing responsibility to evaluate and treat the neurogenic bladder effectively as early as it is detected and over a life span that is approaching that of the normal population.

SUMMARY OF REVIEW OF LITERATURE:

We have summarized the review of literature by the following headings:

1. Anatomy of male vesicourethric unit.
2. Physiology of vesicourethric unit.
3. Abnormal physiology in neurogenic bladder.
4. Structural changes caused by the abnormal function and the typical changes in micturating cystourethrogram due to this.
5. Possible effect on the upper tracts due to these functional and structural changes in the vesicourethric unit in neurogenic bladder.

In this study, we are interested in the quantification of urethral changes and its possible relation if any with the upper tract changes in neurogenic bladder and its comparison with Posterior urethral valves and cystoscopically normal controls.

MATERIAL AND METHODS

Study design: This is a retrospective case control study of male children with a diagnosis of neurogenic bladder, posterior urethral valve and control patients (vesicoureteric reflux), treated for their respective conditions in the Department of Paediatric surgery, Christian Medical College and Hospital(CMC), Vellore, India; between January 2007 to October 2010. The micturating cystourethrograms of all these 197 patients were studied and urethral ratio was calculated in all patients. A detailed study of neurogenic bladder boys with respect to their posterior urethral dilatation was done.

A. Patient groups:

Three groups of patients were studied and urethral ratio was calculated from their Micturating cystourethrograms.

Group	No of patients
Neurogenic bladder (NB)	73
Posterior urethral valve (PUV)	75
Control (VUR)	49

Group 1: Neurogenic bladder.

A total of 73 boys diagnosed as neurogenic bladder with a proven cause (congenital or traumatic) were included in this group.

***Inclusion criteria*:**

a) Boys with congenital neural tube defects in the form of either meningomyelocoele (n=56), lipomeningocoele (n=5), sacral agenesis (n=7) or occult spinal dysraphism with tethered cord syndrome (n=3) as proven by MRI.
b) Boys with acquired cause in the form of spinal trauma alone without pelvic injury (n=2)

Exclusion criteria:

a) Patients with nonneurogenic-neurogenic bladder.
b) Neurogenic bladder associated with anorectal malformation (ARM).
c) Neurogenic bladder secondary to spinal cord tumors.
d) Neurogenic bladder secondary to intracranial pathology, eg, cerebral palsy.

Group 2: Posterior urethral valves :

A total of 75 patients diagnosed and treated for posterior urethral valves, who had a preoperative micturating cystourethrogram done under fluoroscopy in radiology department of CMCH, and were primarily treated in CMC hospital were included in this group.

Exclusion criteria:

a) Boys who underwent fulguration of Posterior urethral valves in other hospitals and then underwent cystourethrogram in CMCH with check cystoscopy and refulguration or any other operative procedure were excluded from the study.
b) Boys who had a micturating cystourethrogram done on the operating table under image intensifier in CMCH were excluded as proper oblique images were not available in those patients.

Group 3: Control patients:

A total of 49 boys who had undergone cystoscopy prior to ureteric reimplantation for primary vesicoureteric reflux were taken as control patients.

Inclusion criteria:

Boys who underwent cystoscopy for primary VUR and on cystoscopy found to have a normal urethra and bladder.

***Exclusion criteria**:*

Patients who had any abnormality in the urethra or trabeculation in the bladder wall on cystoscopy were excluded.

Note that cystoscopy rather than radiology was used as criterion for normality to avoid a radiological bias.

B. Calculation of urethral ratio in micturating cystourethrogram:

The micturating cystourethrograms (MCU) were studied retrospectively and urethral ratio was calculated in all 197 patients. The MCU's were performed under fluoroscopy with both the paediatric surgeon and the radiologist present during the study.

Definition of urethral ratio (UR):

We defined urethral ratio (UR) as the diameter of the posterior urethra (PU) divided by the diameter of the anterior urethra (AU), measured during the voiding phase, on an oblique film of a micturating cystourethrogram

Measurement of urethral ratio [1] [2]

The diameter of the posterior urethra was measured transversely at a point halfway between the bladder neck and the distal end of the membranous urethra. The diameter of the anterior urethra was measured as a transverse diameter at the point of maximum distension in the bulbar urethra .All the measurements were done on a voiding

film, in oblique position without a catheter. Both measurements were taken on the same film and then a urethral ratio was calculated.

C. Additional data for neurogenic bladder patients (n=73 boys):

Since the focus of this study is mainly on neurogenic bladder, additional data was collected for these 73 boys.

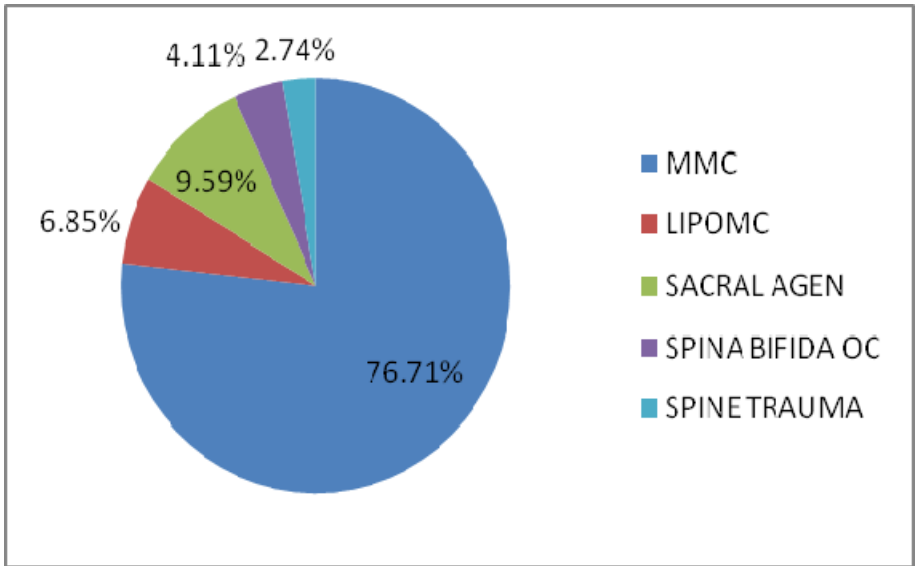
1) Base line patient information:

i. Age: Boys with neurogenic bladder between the age group of 5 months to 15 years of age were included.

Group	Age(year)	No. of patients (n=73)	%
Infancy	< =1	10	13.7
Preschool	2-5	27	36.99
Primary school	6-10	24	32.88
High school	11-15	12	16.44

ii. Etiology of NB: The cause of neurogenic bladder was studied only congenital neural tube defect patients excluding ARM were taken in the study. Two cases of spinal trauma presenting as neurogenic bladder were also included.

Cause of NB	No. of patients	%
Meningomyelocoele	56	76.7
Lipomeningocoele	5	6.85
Sacral agenesis	7	9.59
Spina bifida occulta with tethered cord	3	4.11
Spinal trauma	2	2.74



2) Radiological investigations:

i) Micturating cystourethrograms (MCU) –

MCU's were studied with respect to the following parameters-

a) Bladder trabeculation

b) Vesicoureteric reflux

c) Posterior urethral dilatation.

NB Patients	**No. of patients**		
VUR present	20	Unilateral	12
		Bilateral	8
VUR absent	53		

ii) Ultrasonography (USG) :

The following parameters were studied on USG in each patient-

a) Upper tract changes in the form of hydroureteronephrosis, and whether unilateral or bilateral.

b) Bladder thickening / trabeculations.

Upper tract changes (HUN)	No of patients N=73	%
Present (unilateral/bilateral)	21	28.76
Absent	52	71.24

21 boys (28.76 %) with NB, had upper tract changes in the form of hydroureteronephrosis at presentation, while 52 (71.24 %) had normal upper tracts at presentation. Statistical correlation between upper tract changes and urethral ratio was calculated.

***3) Cystometrogram (CMG*) -**

CMGs if available were studied and any statistical correlation between CMG finding and urethral ratio was assessed. A total of 22 patients underwent CMG.

Bladder Compliance	No. of patients (N=22)	%
Good	5	22.72
Poor	17	77.28

4) Treatment:

All neurogenic bladder patients were studied for the mode of treatment i.e medical /surgical. Medical management included mainly CIC and anticholinergic drugs (oxybutinin). Few patients required surgery in the form of either diversion (vesicostomy/ureterostomy) or bladder augmentation.

Treatment	No of NB patients (n=73)	% of NB patients
Medical	63	86.3
Surgical	10	13.7

D. Statistical Methods:

Data entry was undertaken by a single investigator using Microsoft excel. Data analysis was done using Statistical Package for Social Version 15.Comparison of urethral ratios between three groups of patients was made using multiple comparisons (ANOVA) and statistical difference sought. T-test was used to assess if there is any statistical difference in the urethral ratio in different age groups, patients with or without upper tract changes and compliance on CMG.

RESULTS AND ANALYSIS

1. Patient Group Vs Urethral ratio (UR):

Micturating cystourethrograms of all patients were studied and Urethral ratio was calculated in all three groups.

Patient group	Mean UR	Range	Std. Deviation	95% Confidence Interval for Mean		ANOVA p value
				Lower Bound	Upper Bound	
Controls (n=49)	1.28	0.5-3.2	0.47	1.15	1.42	0.000
Neurogenic bladder (n=73)	2.92	0.8-8.4	1.68	2.53	3.31	
Posterior urethral valves (n=75)	5.12	1.06-13.9	2.85	4.46	5.78	

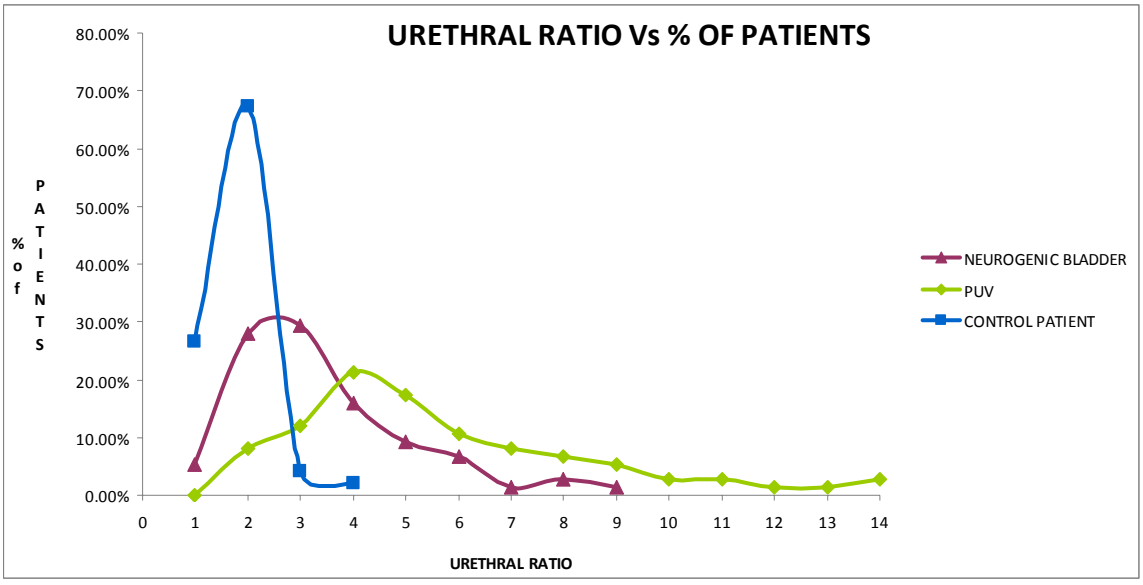
The mean urethral ratio in control patients was 1.28 (SD 0.47), with a range of 0.5 to 3.2. In neurogenic bladder mean (SD) was 2.92 (1.68), with a range of 0.8 to 8.4 and in Posterior urethral valves it was 5.12 (2.85) and a range of 1.06 to 13.9.

Multiple Comparisons

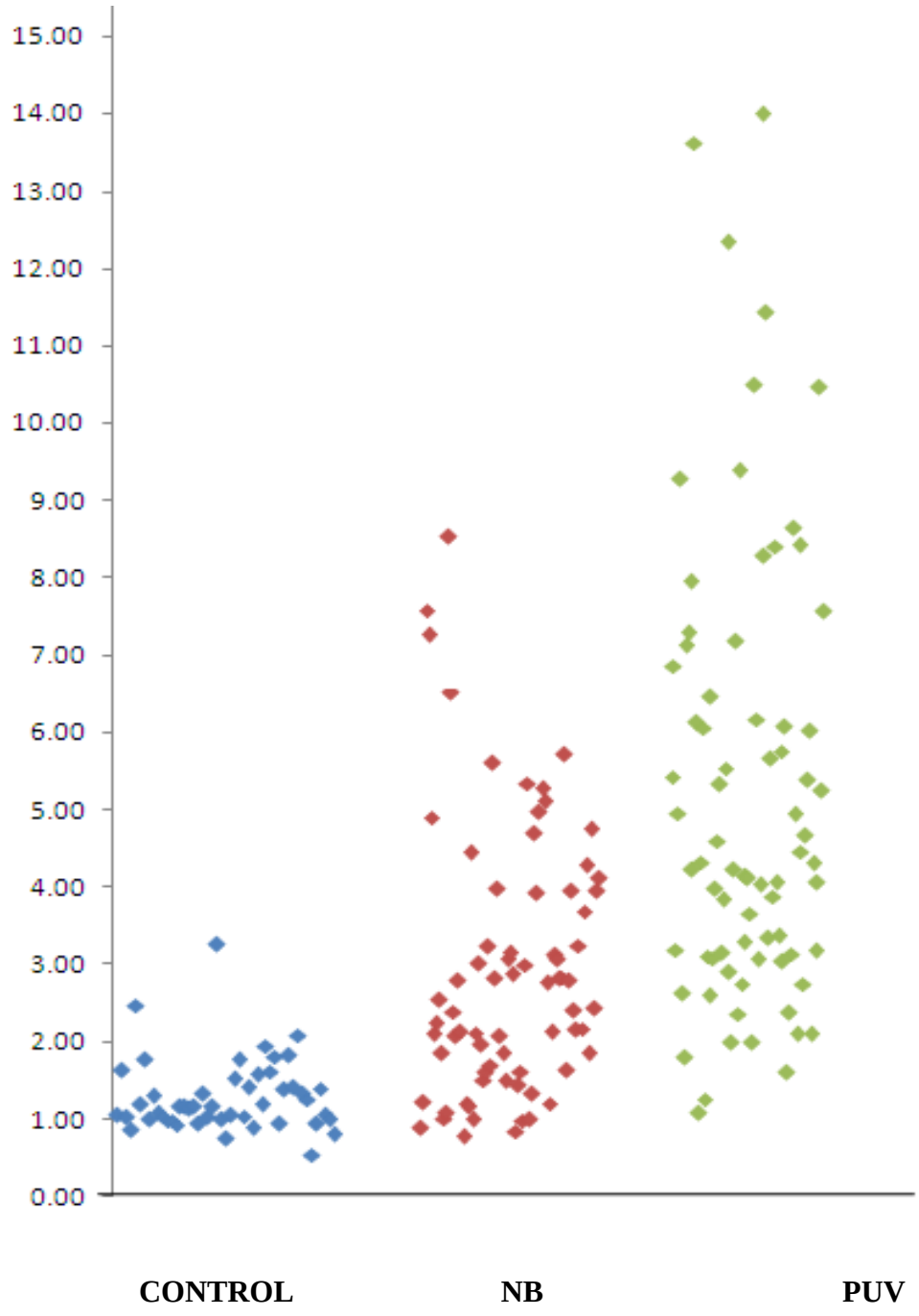
(I) GROUPS	(J) GROUPS	Mean Difference (I-J)	Sig.	95% Confidence Interval	
				Lower Bound	Upper Bound
1	2	-1.594	.000	-2.511	-.677
	3	-3.836	.000	-4.748	-2.924
2	1	1.594	.000	.677	2.511
	3	-2.241	.000	-3.057	-1.425
3	1	3.836	.000	2.924	4.748
	2	2.241	.000	1.425	3.057

On comparing the urethral ratio between all three groups of patients, the urethral ratio in neurogenic bladder patients was significantly higher than controls ($p<0.05$) and lower than PUV patients, similarly, the urethral ratio in PUV patients was significantly higher than both the other groups ($p<0.05$)

Graph showing distribution of urethral ratio in all three groups expressed as percentage of the population.



Graph Showing distribution of Urethral Ratio in Each Group:



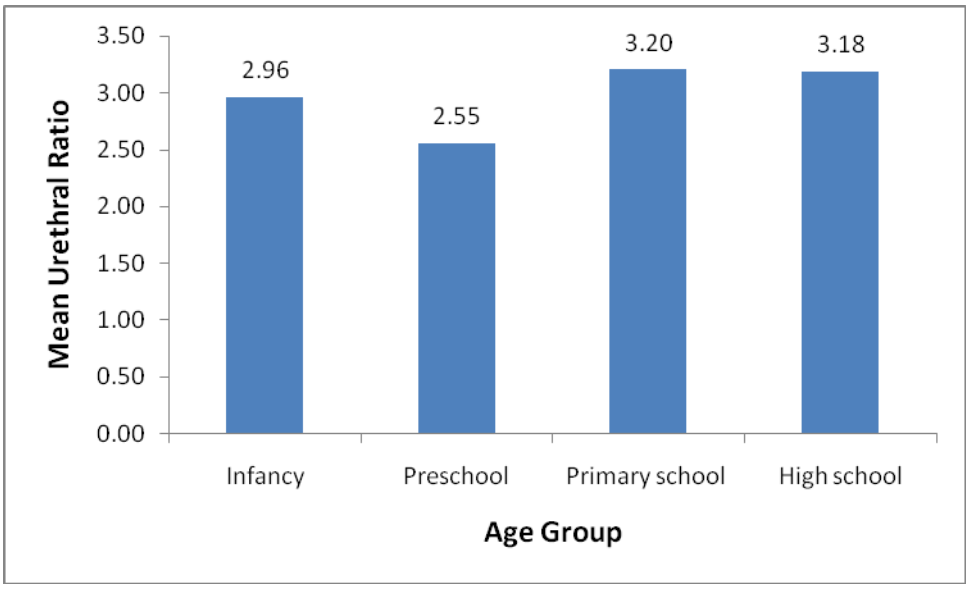
Though the mean urethral ratio was different in each of the group, there was some overlap in the U.R between the control group and neurogenic bladder and also between neurogenic bladder and PUV group.

2. Age at presentation Vs Urethral ratio:

Age Group(years) N=73	Mean UR	Range	S.D	95% Confidence Interval for Mean		P value
				lower bound	Upper bound	
Infancy (<=1)	2.96	0.8-5.65	1.71	1.73	4.18	0.529
Preschool (2-5)	2.55	0.9-7.5	1.50	1.95	3.14	
Primary school (6-10)	3.20	0.86-8.44	1.74	2.46	3.94	
High school (11-15)	3.18	1.1-7.18	1.97	1.92	4.43	

The mean age of neurogenic bladder patients at presentation was 6.02. The mean urethral ratio was highest 3.2 (SD 1.74) in the primary school age group, and lowest in the 2 to 5 age group 2.55 (SD 1.50). There was no correlation between age at presentation with neurogenic bladder and the urethral ratio (p=0.529)

Age group Vs Mean UR:



Multiple Comparisons

(I) Age_rec 1	(J) Age_rec 1	Mean Difference (I-J)	Sig.	95% Confidence Interval	
				Lower Bound	Upper Bound
< =1 yr	2-5 yr	.405	1.000	-1.299	2.111
	6-10 yr	-.245	1.000	-1.979	1.488
	11-15 yr	-.226	1.000	-2.198	1.746
2-5 yr	<=1 yr	-.405	1.000	-2.111	1.299
	6-10 yr	-.651	1.000	-1.943	.641
	11-15 yr	-.631	1.000	-2.230	.966
6-10 yr	<=1 yr	.245	1.000	-1.48	1.979
	2-5 yr	.651	1.000	-.641	1.943
	11-15 yr	.019	1.000	-1.609	1.648
>11 yr	<=1 yr	.226	1.000	-1.746	2.198
	2-5 yr	.631	1.000	-.966	2.230
	6-10 yr	-.019	1.000	-1.648	1.609

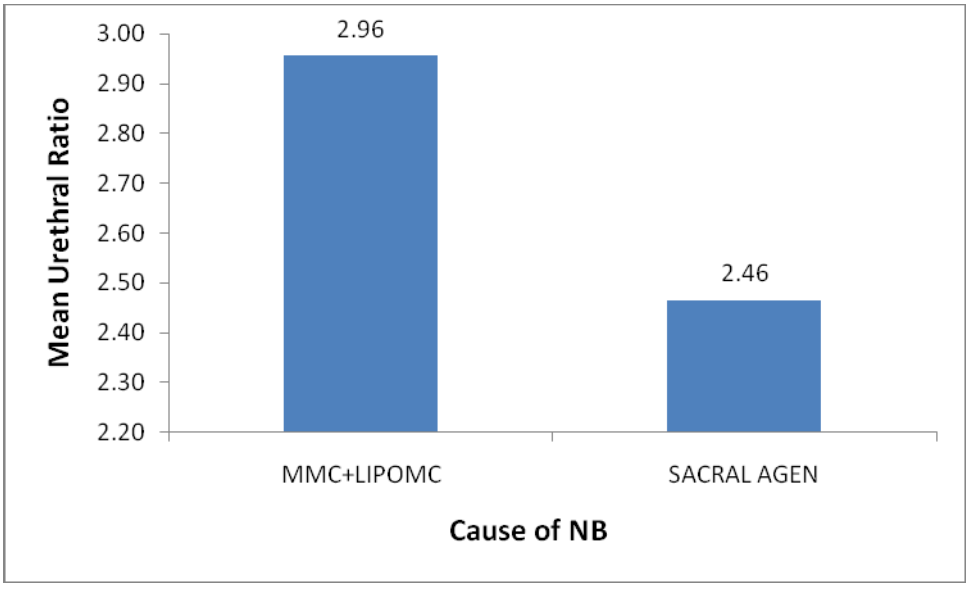
On comparing different age groups, no statistical correlation was found between age at presentation with neurogenic bladder and urethral ratio ($p>0.05$)

3. Cause of neurogenic bladder Vs urethral ratio:

Cause of NB n=68	Mean UR	S.D	95% confidence interval of the difference		P value
			Lower	Upper	
MMC+LipoMc n=61	2.96	1.78	-0.88	1.86	0.477
Sacral agenesis n=7	2.46	0.90	-0.39	1.38	

The mean urethral ratio in MMC+Lipomeningocoele group was 2.96 while in sacral agenesis patients it was 2.46. However, in patients with MMC and lipomeningocoele as compared with sacral agenesis, no statistically significant difference in urethral ratio was found.

Cause of NB Vs Mean UR:

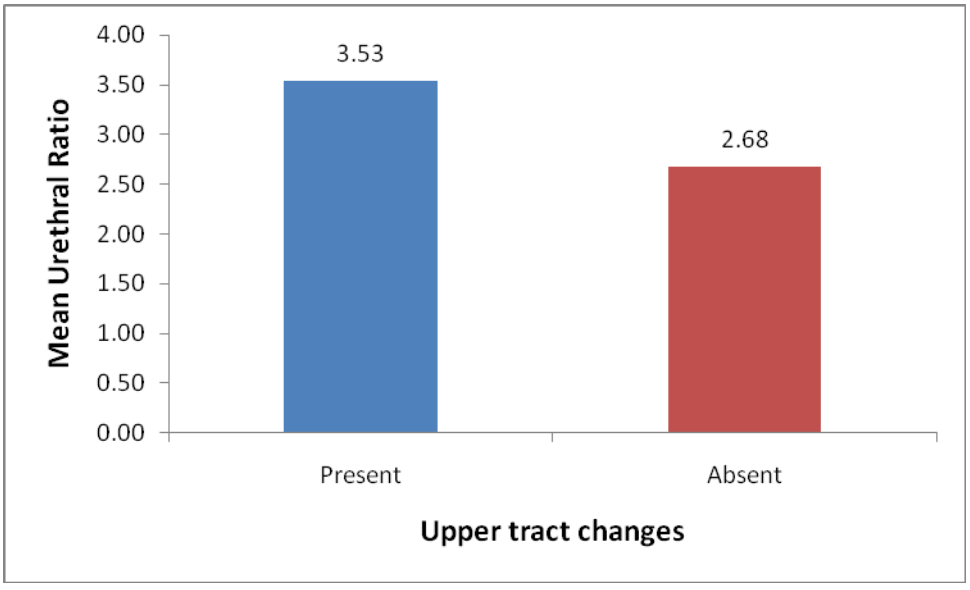


4. Upper tract changes Vs Urethral ratio:

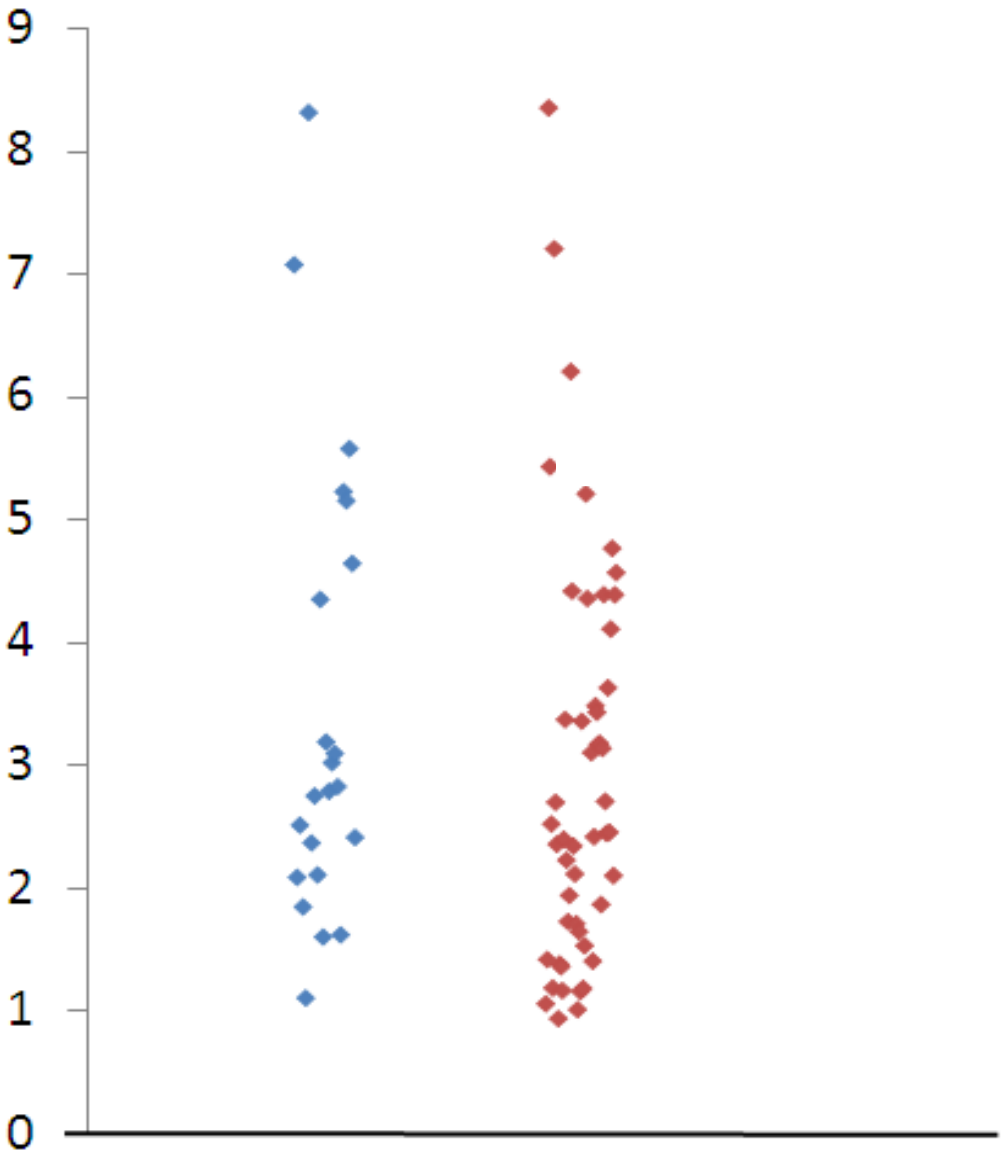
Upper tract changes (HUN) N=73	Mean UR	S.D	S.E	P value
Present(n=21)	3.53	1.92	0.420	0.049
Absent(n=52)	2.67	1.53	0.212	

In boys who had neurogenic bladder with upper tract changes, the mean (SD) urethral ratio was 3.53(1.92), while in those without upper tract changes, it was 2.67 (1.53). Thus, the urethral ratio in boys with neurogenic bladder who had upper tract changes (in the form of hydroureteronephrosis on USG) was significantly higher than in those boys without upper tract changes ($p<0.05$)

Upper tract changes Vs Mean UR:



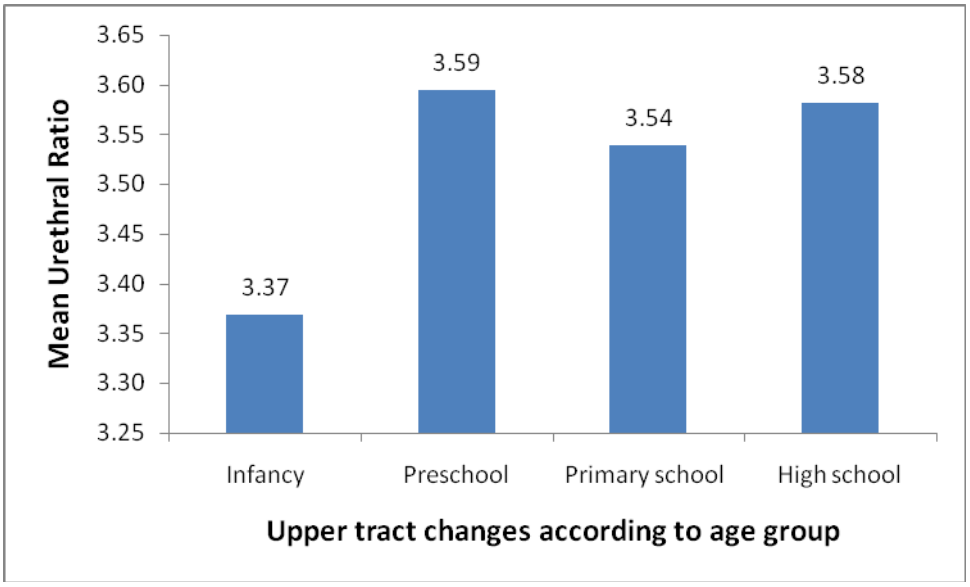
Graph showing distribution of UR Vs Upper tract changes in neurogenic bladder:



5. Upper tract changes according to age group Vs mean UR:

Upper tract changes in Age group N=21	Mean UR	S.D	95% Confidence Interval for Mean		Range		*p* value
			Lower Bound	Upper Bound	Min	Max	
Infancy (n=3)	3.37	2.07	-1.7835	8.5035	1.62	5.65	0.999
Preschool (n=2)	3.59	1.14	-6.7020	13.8820	2.78	4.40	
Primary school (n=9)	3.54	2.09	1.9204	5.1396	1.86	8.44	
High school (n=7)	3.58	2.21	1.5324	5.6219	1.10	7.18	

Upper tract changes according to age group Vs mean Urethral Ratio:



In patients with upper tract changes, the mean urethral ratio (3.59) was highest in the preschool age group, and lowest in infancy (3.37)

Multiple Comparisons

(I) Age_rec	(J) Age_rec	Mean Difference (I-J)	Sig.	95% Confidence Interval	
				Lower Bound	Upper Bound
<=1 yr	**2-5 yr**	-.23000	1.000	-5.9246	5.4646
	6-10 yr	-.17000	1.000	-4.3287	3.9887
	11-15 yr	-.21714	1.000	-4.5218	4.0876
2-5 yr	**<=1 yr**	.23000	1.000	-5.4646	5.9246
	6-10 yr	.06000	1.000	-4.8165	4.9365
	11-15yr	.01286	1.000	-4.9888	5.0145
6-10 yr	**<=1 yr**	.17000	1.000	-3.9887	4.3287
	2-5 yr	-.06000	1.000	-4.9365	4.8165
	11-15 yr	-.04714	1.000	-3.1909	3.0966
11-15 yr	**<=1 yr**	.21714	1.000	-4.0876	4.5218
	2-5 yr	-.01286	1.000	-5.0145	4.9888
	6-10 yr	.04714	1.000	-3.0966	3.1909

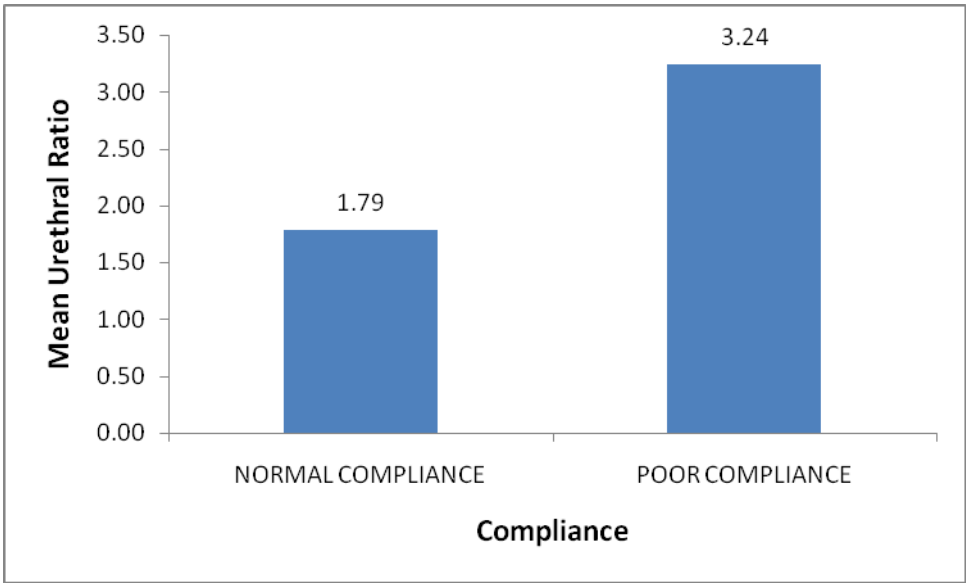
On comparison of the urethral ratio in boys with neurogenic bladder having upper tract changes (hydroureteronephrosis on USG) in the different age groups, no statistically significant difference was found between the urethral ratio and the age group.

6. Bladder compliance on CMG Vs Urethral ratio:

COMPLIANCE	MEAN UR	S.D	95% confidence interval of the difference		*p* Value
			Lower	Upper	
NORMAL (n=5)	1.79	1.08	-2.71	-0.19	0.026
POOR (n=17)	3.24	1.21	-2.78	-0.12	

The mean (SD) urethral ratio in neurogenic bladder patients with poor compliance on CMG was 3.24 (SD 1.21) which was significantly higher ($p<0.05$) than in those with normal compliance 1.79 (SD 1.08).

Compliance Vs Mean Urethral Ratio:



SUMMARY OF RESULTS:

1. On comparing the urethral ratio between all three groups of patients, the mean urethral ratio in neurogenic bladder (2.92) was significantly higher than controls (1.28) ($p<0.05$) and lower than PUV patients, similarly, the urethral ratio in PUV patients (5.12) was significantly higher than both the other groups ($p<0.05$).

2. There was no statistically significant difference found between age at presentation with neurogenic bladder and the urethral ratio.

3. In patients with meningomyelocoele and lipomeningocoele as compared with sacral agenesis, no statistically significant difference in urethral ratio was found.

4. In neurogenic bladder patients with upper tract changes, the mean (SD) urethral ratio was 3.53(SD 1.92), while in those without upper tract changes, it was 2.67(SD1.53). Thus, the urethral ratio was significantly higher in neurogenic bladder with upper tract changes than in those without upper tract changes ($p<0.05$)

5. The mean (SD) urethral ratio in neurogenic bladder patients with poor compliance on CMG was 3.24(SD1.21) which was significantly higher ($p<0.05$) than in those with normal compliance 1.79(SD 1.21).

DISCUSSION

The majority of patients with neurogenic bladder are born with structurally normal upper tracts, unlike patients with posterior urethral valves. Thus much of the renal damage is acquired after birth, except in some cases of sacral agenesis where structural problems such as solitary kidney may considerably increase the risk of renal failure. In neurogenic bladder, there is a behavioural problem with the bladder and the urethral sphincter –the vesicouretheric unit. Proper behaviour expected of the vesicouretheric unit is a quiet bladder during storage and a quiet sphincter during voiding. A bladder which is 'active' during storage (neurogenic detrusor overactivity) and a sphincter that is active during voiding (detrusor sphincter dyssynergia) is the most common and most dangerous type of problem seen in neurogenic bladder. Over a period of time, or sometimes very quickly, this leads to bladder muscle hypertrophy, collagen deposition, high pressure storage and secondary vesicoureteric reflux -much the picture of Posterior urethral valves. Hence it is imperative to distinguish the neurogenic bladder and especially non-neurogenic neurogenic bladder from PUV, as the management of all these conditions differs.

The micturating cystourethrogram forms the basic investigation in PUV, neurogenic bladder and vesicoureteric reflux (± dysfunctional voiding), however there are some limitations of this study, as the bladder, posterior urethra and upper tracts may appear identical in all these conditions, and hence may cause diagnostic confusion and inappropriate management. The posterior urethra has a tremendous capacity to stretch with increased voiding pressure due to its unique histological and anatomical configuration and hence a dilated posterior urethra may be found

whenever there is disturbance in the vesicourethric unit. In this study we have compared this dilatation of posterior urethra on micturating cystourethrograms of neurogenic bladder boys, Posterior urethral valves and vesicoureteric reflux with a cystoscopically normal urethra (control group).

O.Bani Hani from Australia and Gupta et al and Menon et al from India have published studies on PUV patients where the authors have quantified the posterior urethra to bulbar urethral ratio on micturating cystourethrograms , pre and post fulguration, and concluded that reduction in the urethral ratio in the post-fulguration MCU can be considered as an objective evidence of adequacy of valve fulguration [1,2,3]. We have utilized this calculation of urethral ratio in patients with neurogenic bladder and compared it with PUVs and controls.

This present study consists of 73 neurogenic bladder boys, 75 PUV patients and 49 control patients. In choosing the cases of neurogenic bladder we have taken only those boys with proven neurological lesion in spinal cord while nonneurogenic neurogenic bladder cases were excluded. Since MCU and cystourethroscopy is not generally done in normal boys, as Controls we have taken boys with primary vesicoureteric reflux who underwent MCU and cystoscopy prior to reimplantation, ruling out cases with evidence of bladder trabeculation. The cystourethroscopy rather than radiology was taken as guideline for defining normality in these cases to avoid bias. We have calculated the urethral ratio on the MCU in all these three groups of patients and found that the urethral ratio in neurogenic bladder patients was significantly higher (mean 2.92, SD 1.68) than in the control group (mean1.28, SD

0.47), but lower than that in the posterior urethral valves (Mean5.12, SD 2.85), but still all three groups had a overlap of their ratios.

In our study we have attempted to study whether the dilatation of posterior urethra increases according to the age of the child at presentation with neurogenic bladder. Though the mean urethral ratio was highest (3.2) in the primary school age group(6-10years), and lowest (2.55) in the preschool (2 to 5 years) age group no correlation between age at presentation with neurogenic bladder and the urethral ratio (p=0.529) was found. It is a known fact that the neurourologic lesion in myelodysplasia is a dynamic disease process with changes taking place throughout childhood, [38-40] especially in early infancy, [30] and then at puberty, [41] when the linear growth rate accelerates again. Fifteen to 20 percent of newborns have an abnormal urinary tract on radiologic examination when first evaluated [42]. Without proper management, urinary tract infections and elevated bladder pressures with secondary bladder wall changes may cause upper urinary tract deterioration within 3 years in upto 58% of patients [29].

We have compared the urethral ratio in two groups of neurogenic bladder patients, first group was meningomyelocoele and lipomeningocoele (UR=2.96) taken together and other group was sacral agenesis (UR=2.46).Our study does not show any statistically significant difference in the dilatation of posterior urethra in these two groups of patients, however the number of sacral agenesis (N=7) patients are far fewer than MMC+Lipomeningocoele (N=61). Although prognosis is believed to be correlated with the type and severity of neural tube defects (NTD), and in general, MMC is considered more severe than sacral agenesis or caudal regression syndrome

(CRS), there are no studies in the literature comparing the long-term urologic outcome in different populations of neural tube defects. Studies have shown that myelomeningocele accounts for more than 90% of all open spinal dysraphic states [43] and almost 100% of them have some degree of neurologic impairment. Sacral agenesis is not always associated with neuropathic bladder (NB) but can present as different urologic patterns with a great variability. Torrea et al have compared urologic outcome of patients with CRS as compared with MMC. They concluded that 61% of patients with CRS and 98% of those with MMC had NB, and the functional prognosis of renal function was not better in those with CRS than in MMC [44].

In this study we have analysed whether amount of posterior urethral dilatation has any relation to the upper tract changes in neurogenic bladder. In those with upper tract changes (as shown by hydroureteronephrosis on USG), the mean urethral ratio was 3.53(SD1.92), while in those without upper tract changes it was 2.67 (SD1.53). Thus, the urethral ratio was significantly higher in those with upper tract changes ($p<0.05$) and hence quantification of posterior urethral dilatation can be correlated with hydronephrosis .Past studies have shown that serum creatinine values and ultrasound imaging do not provide a sensitive measure of early renal injury, as changes may arise only after significant renal damage. Therefore, can we consider severity of posterior urethral dilatation in DESD as an indicator of progressive renal deterioration? Expectant treatment has revealed that infants with bladder outlet obstruction in the form of DESD are at considerable risk for urinary tract deterioration. Hence more the severity of bladder outlet obstruction, more will be the dilatation of posterior urethra and more will be the upper tract deterioration. It

is a proven fact that elevated detrusor pressures and recurrent urinary tract infections are the primary risk factors for renal deterioration and may be prevented by appropriate care of the lower tract [16, 45].

Although different aspects of the urologic treatment of children with myelodysplasia may be emphasized at different points in their lives (such as continence, sexual function, ease of catheterization), the ultimate challenge is to maintain normal upper urinary tracts by preventing renal injury.

We have also analysed whether the neurogenic bladder boys, who present at a later age have more chances of upper tract deterioration as compared to those who present early, and the relation of posterior urethral dilatation in these patients. However no statistical correlation was found between these parameters. We have not found any studies in the literature stating this correlation.

We routinely perform cystometrograms in neurogenic bladder cases at first visit and subsequently, to see for compliance and to rule out DESD. In this study we have noticed that the urethral dilatation was significantly higher in those with poorly compliant bladder (Mean UR 3.24), and less in those with good compliance (Mean UR 1.79). We feel that as the bladder outlet obstruction increases the posterior urethra dilates more and the compliance decreases, and this has been proved by our study. Studies have shown that sequential urodynamic testing on a yearly basis beginning in the newborn period and continuing until the child is 5 years old provides a means of carefully monitoring these children to detect signs of change. Approximately 10% of newborns with myelomeningocele exhibit no abnormality on urodynamic testing [46]. Most children who undergo changes tend to do so in the first 3 years of life [47].

Risk factors such as decreased bladder compliance and high leak point bladder pressure should be recognized early to initiate clean intermittent catheterization, anticholinergic drug treatment and prophylactic antibiotics rational therapy with CIC and anticholinergics depends on proper diagnostic tools. Undoubtedly, cystometrogram (CMG) is a valuable tool in the follow up of treatment, especially in regard to bladder compliance.

In the present study we wish to emphasize that the posterior urethral dilatation exists not only in posterior urethral valves but also in many cases of neurogenic bladder, especially in those at risk for upper tract changes. Though as compared to PUV, posterior urethral dilatation is less in neurogenic bladder, it definitely has a overlap with PUV. Posterior urethral dialatation does not seem to have relation with age of the child, but it definitely shows correlation with renal damage and bladder compliance and thus is a diagnostic and prognostically important finding in MCU.As a extrapolation of the results obtained from our study, it is important to recognize the posterior urethral dilatation in cases of non-neurogenic neurogenic bladder who have no other stigmata of neurological disease and thus may be confused with PUV. The quantification of urethral ratios in our study should help in making this important differentiation.

CONCLUSION

1. Posterior urethra is significantly dilated in boys with neurogenic bladder compared with normal controls.
2. The posterior urethral dilatation in neurogenic bladder is less than that in the posterior urethral valves, although there is a overlap in both groups.
3. Boys with neurogenic bladder and upper tract changes showed significantly higher urethral ratio as compared with those without upper tract changes.
4. Neurogenic bladder with poor compliance has significantly higher urethral ratio than those with normal compliance on cystometrogram.

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ABBREVATIONS

PUV – Posterior urethral valves

NB – Neurogenic bladder

VUR – Vesicoureteric reflux

MMC – Meningomyelocoele

NNNB – Nonneurogenic neurogenic bladder

MCU – Micturating Cystourethrogram

DESD – Detrusor external sphincter dysfunction

CIC – Clean intermittent catheterization.

UR – Urethral ratio

AU – Anterior urethra

PU – Posterior urethra

CMG – Cystometrogram

LipoMC – Lipomeningocoele

NTD – Neural tube defect

USG – Ultrasonography

. KEY TO MASTERSHEET

Cause of NB= cause of neurogenic bladder
- 1= Meningomyelocoele
- 2= Lipomeningocoele
- 3= Sacral agenesis
- 4= Spina bifida occulta with tethered cord
- 5= Spine trauma

U.R= URETHRAL RATIO

HN=hydronephrosis on ultrasound
- 1= Present
- 2= Absent

BLD TRAB= Bladder trabeculation
- 1= Present
- 2= Absent

REFLUX
- 1= Present
- 2= Absent

P.U DILN= Posterior urethral dilatation on MCU report.
- 1= Present
- 2= Absent

CREAT=Creatinine

PVR= Postvoid residue

TREATMENT
- 1= CIC
- 2= CIC+Oxybutynin
- 3= CIC+Oxybutynin+Amitryptiline
- 4= Oxybutynin only
- 5= Diversion
- 6= Bladder augmentation+/- Mitrafanoff.

CMG= cystometrogram
- 1= Normal compliance
- 2= Poor compliance.

SR.NO	HOSP NO	NAME	AGE	CAUSE OF NB	U.R	HN	BLD TRAB	REFLUX	PU DILN	CREAT	PVR	TREATMENT	CMG
1	589608C	GEETHA'S BABY	4	1	0.909091	2	1	2	2	0.5	1	1	1
2	244473D	LATHA'S BABY	1	1	1.234615	2	2	2	2	0.4	2	1	
3	242207D	AMIT MATHEW THOMAS	3	2	7.5	2	1	2	2	0.5	1	2	
4	369214D	KAVIYARASU	13	1	7.183099	1	1	2	1	0.7	1	2	
5	199189D	DEBU HALDAR	0.5	1	4.860465	2	2	1	1	0.4	1	1	
6	321693D	MUNNA KUMAR	12	1	2.105263	1	1	1	2	2.4	1	1	
7	083763D	DHANRAJ SARVESH	3	3	2.232143	2	2	2	2	0.5	1	1	
8	338592D	MD.SHAHEED ALAM	6	1	2.538462	1	1	1	2	0.8	1	5	
9	109534C	SATHISH M	6	1	1.861111	1	1	2	1	0.6	1	5	
10	405224C	SUMATHIS BABY	3	1	1.025641	2	2	2	2	0.4	1	1	
11	315567D	TANMAY SAHU	14	2	1.105528	1	1	2	2	0.6	1	2	
12	202545D	YUVRAJ	10	1	8.444444	1	1	2	1	0.8	1	1	
13	833388B	VELMURUGAN	8	1	6.463158	2	1	2	1	0.6	1	1	
14	267157D	RANGARAJ	10	1	2.391304	2	1	2	1	0.5	2	2	
15	235725C	PRABHAVATHI'S BABY	6	1	2.082927	2	2	2	1	0.6	2	6	
16	986509C	RAJU	5	1	2.78022	1	1	1	2	0.6	2	6	
17	019336D	JAMSHEDUDDIN	7	3	2.125984	1	1	2	2	0.5	2	1	
18	815795C	RAJKUMAR	1	1	0.8	2	2	2	2	0.4	2	1	1
19	090466D	AYUSHKUMAR VARMA	2	1	1.20202	2	2	2	2	0.4	1	3	
20	099149D	MALAY BERA	1	1	1.18239	2	2	2	2	0.4	1	2	
21	822076C	AKASH SARKAR	2	1	4.409091	1	1	1	1	0.7	2	6	
22	034947C	GEETHA'S BABY	5	1	1.006623	2	2	2	2	0.5	2	4	2
23	024609D	MUKESHKUMAR	8	1	2.120482	2	1	2	1	0.7	1	1	
24	962196C	LIYAKAT ALI	8	1	3	2	1	2	1	0.5	1	2	
25	896802C	KONMAN	2	1	1.966292	2	2	2	2	0.4	2	1	
26	015891D	KRISHNA SUMANTH	15	1	1.51375	2	2	2	2	0.6	2	1	2
27	121664D	VINAYAK	12	1	1.611289	1	1	1	2	0.4	1	1	
28	036123D	VINEET VICTOR	6	1	3.227513	1	1	2	1	0.5	2	2	2
29	765698C	SUDIP DAS	2.5	1	1.706093	2	2	2	2	0.4	1	2	
30	522749B	VIGNESH	11	1	5.560811	2	2	2	1	0.6	2	6	
31	838989C	SELVI'S BABY	1	4	2.818182	1	1	1	1	0.5	2	1	
32	965407C	SWAGATAM DAS MANDAL	2	1	**3.945**	2	1	2	1	0.4	1	2	2
33	158360D	TARNI SEN MANDAL	12	1	2.07029	2	2	2	2	0.5	1	1	
34	426226C	SHAHEED BANDWA	3.5	1	1.864489	2	2	2	1	0.4	1	1	
35	927255C	SURYA S	5	1	1.497485	2	2	2	2	0.5	2	2	
36	436775D	ANANDA GIRI	8	1	3.055556	1	1	1	+	0.6	2	6	
37	390506D	BHABATOSH	13	3	3.132552	1	1	1	1	1.6	2	6	2
38	453779D	DEVA	9	1	2.857143	1	1	2	1	1.4	2	1	2
39	458458D	ESTIAQUE.A	8	1	0.866667	2	1	1	2	0.6	2	2	
40	949577C	GAUTHAM	4	4	1.441176	2	1	1	2	0.4	1	5	2
41	239030D	JHUMA BAGACHI'S BABY	1	1	1.628788	1	2	1	1	0.4	2	5	
42	406579D	KUMARAN	2	3	1	2	1	2	2	0.4	2	1	
43	384236D	MAYANK SHARMA	3	1	2.988372	2	2	2	1	0.5	2	2	
44	652058B	MOHD SAINAN	6	1	5.302419	1	1	2	1	0.5	1	2	
45	564805D	NILESH KUMAR OJHA	2	2	1.022222	2	1	2	2	0.4	1	1	
46	555344D	PRITAM C	8	1	1.337079	2	2	2	2	0.7	1	1	1
47	171030C	RANJITH KUMAR	8	1	4.66055	2	1	2	1	0.6	1	1	2
48	3970686C	SABITRIDEVI'S BABY	5	1	3.891176	2	1	2	1	0.6	1	2	2
49	481149D	SAMRAT GHOSH	2	1	4.929293	2	1	2	1	0.4	1	2	
50	598707D	SAMU	14	1	5.226415	1	1	2	1	0.6	1	2	2
51	386855D	SANTHANA KUMAR	9	1	5.075	2	1	2	1	0.5	2	2	
52	426226C	SHAHIL BURNWAL	5	1	2.757396	2	2	2	2	0.4	1	2	
53	491208D	SHARIERAAHAMED	8	1	1.223404	2	1	2	2	0.6	1	2	
54	517658D	SHUBHAM KUMAR	14	1	2.137931	2	1	2	1	0.6	1	4	
55	499755D	SUDIP KUMAR	4	2	3.10101	2	1	2	1	0.6	1	4	1
56	440086D	SUJAL GUPTA	1	2	3.053846	2	2	2	1	0.5	2	1	
57	444227D	SUSHANT SHURAV	5	1	2.817647	2	2	1	2	0.5	1	2	
58	396904D	TANMOY SAHA	7	5	2.824818	2	2	2	1	0.6	1	4	
59	955364C	USHA'S BABY	0.5	1	5.658824	1	2	2	+	0.5	2	2	
60	512448D	VIJAY SARKAR RAMUL	4	1	1.64	2	2	1	2	0.4	1	2	
61	029535D	VIJAYALAKSHMI'S BABY	2	1	2.7875	2	1	1	1	0.5	1	1	1
62	743963C	ADRITA DEB	8	3	3.919753	2	1	2	1	0.5	2	2	2
63	642833D	DEPARPAN MUKHERJEE	2	3	2.398333	2	1	2	2	0.5	2	4	2
64	653744D	DINESH KUMAR YADAV	10	1	2.163636	2	1	1	1	0.6	2	3	
65	433405C	JAMES	6	1	3.234043	2	1	2	1	0.5	2	1	2
66	639592D	LALPEKHLUA	2.5	1	2.171429	2	1	1	2	0.5	2	2	2
67	683355D	PRIYANSHU	7	1	3.666667	2	1	2	1	0.6	1	1	2
68	743295D	RAGHAVI NATHAN	0.8	4	4.26	2	2	2	1	0.5	1	1	
69	696075D	SABARINATHAN T	12	1	1.852174	2	1	1	2	0.6	2	1	
70	720259D	SHIVSAGAR SHAH	14	5	4.707071	1	1	2	1	1	1	2	2
71	683878D	TORIQUL ISLAM	8	3	2.437403	1	2	1	2	0.6	2	2	
72	499241C	DEEPA'S BABY	5	1	3.918083	2	1	1	2	0.7	2	1	
73	819651D	ARNAB HOTA	1	1	4.081244	2	2	2	2	0.4	2	3	2

NEUROGENIC BLADDER				PUV			CONTROL PATIENT (VUR)		
SR.NO	NAME	HOSP NO	RATIO	NAME	HOSP NO	RATIO	NAME	HOSP NO	RATIO
1	GEETHA'S BABY	589608C	0.91	DEBASHISH SHAW	129817D	5.38	ADITYA KUMAR	217415D	1.06
2	LATHA'S BABY	244473D	1.23	DISANT PRADHAN	003841D	6.81	AJAY PAUL	733425C	1.63
3	AMIT MATHEW THOMAS	242207D	7.50	DHANUSH	806392D	3.16	AMAN RAJ	782802D	1.02
4	KAVIYARASU	369214D	7.18	AMAN VERMA	169767D	4.92	ARIB NOOR FAROOQI	287000D	0.85
5	DEBU HALDAR	199189D	4.86	AYAN KONAR	790466C	9.22	ARPIT RAJ	458298D	2.47
6	MUNNA KUMAR	321693D	2.11	MANOMAY CHAKRABORTY	509241C	2.59	AYUSH KEDIA	386337D	1.18
7	DHANRAJ SARVESH	083763D	2.23	SABARNA KARMAKAR	798701D	1.77	BIKRAM RIT	331021D	1.76
8	MD.SHAHEED ALAM	338592D	2.54	RAMANA	915292C	7.07	BILOMBO BASAK	445039D	1.00
9	SATHISH M	109534C	1.86	PARUEZ BADSHA	087170D	7.23	BIVEK KUMAR	704128D	1.30
10	SUMATHIS BABY	405224C	1.03	PRADEEP	961355C	7.89	CHITRAS BABY	629131D	1.08
11	TANMAY SAHU	315567D	1.11	NITYA BERA	104512D	4.19	DEEPAK M	210904D	1.03
12	YUVRAJ	202545D	8.44	NITHISH KUMAR	078495D	13.53	DHEMAN ROY	254057D	0.98
13	VELMURUGAN	833388B	6.46	YASWANTH	033455D	6.08	DHINESH KUMAR	756190C	0.98
14	RANGARAJ	267157D	2.39	VINAYAGAM	630448C	1.06	FERODES BEGAM'S BABY	859140C	0.92
15	PRABHAVATHI'S BABY	235725C	2.08	USHAS BABY	955364C	4.29	GAURAV MASTER	594328D	1.17
16	RAJU	986509C	2.78	SHRIKANT JANA	989392C	6.01	HEENAKAR THAKUR	490448D	1.15
17	JAMSHEDUDDIN	019336D	2.13	SREEJAN KESARI	168445D	1.23	IBAN KITLANG	298651D	1.13
18	RAJKUMAR	815795C	0.80	SUMAN SUTRADHAR	156299D	3.08	JASWANT S	472637D	1.17
19	AYUSHKUMAR VARMA	090466D	1.20	SHAMEERA BANUS BABY	073037D	2.56	KUTU	664852C	0.94
20	MALAY BERA	099149D	1.18	SUBRAJIT DEB	077900D	6.40	KRISHNENDU PAL	393686D	1.33
21	AKASH SARKAR	822076C	4.41	ASHOK RANJAN S	885484C	3.05	LACKI	116793D	1.03
22	GEETHA'S BABY	034947C	1.01	ANBU D	238246D	3.94	MEERAJ MONDAL	535880D	1.16
23	MUKESHKUMAR	024609D	2.12	ANJALI DEVIS BABY	885965C	4.56	MOHD AMASH	983190C	3.25
24	LIYAKAT ALI	962196C	3.00	BABU	245960D	5.28	MOHD JASEEM	181991D	1.00
25	KONMAN	896802C	1.97	BISHAL GUPTA	162851D	3.11	MOHD SHAHEEN	475612D	0.74
26	KRISHNA SUMANTH	015891D	1.51	CHARAN K	562897C	3.81	MRIGANKA GUHA	306039C	1.05
27	VINAYAK	121664D	1.61	DEVIS BABY	233409D	5.49	NANCY'S BABY	144633D	1.52
28	VINEET VICTOR	036123D	3.23	IYANNER	308175D	12.26	PRASANTH	903316C	1.77
29	SUDIP DAS	765698C	1.71	INZAMAMUL HAQUE	186789D	2.88	PRETIVIRAJ	661842C	1.04
30	VIGNESH	522749B	5.56	JASIMUDDIN AHAMED	221705D	1.97	PUNITHA'S BABY	194272D	1.41
31	SELVI'S BABY	838989C	2.82	LOGNO SAHA	234192D	4.19	RAJDEEP PRAMANIK	709571D	0.89
32	SWAGATAM DAS MANDAL	965407C	3.95	MOHITH NAYAK	368276D	7.12	RAJDITYA GHORAI	299781D	1.58
33	TARNI SEN MANDAL	158360D	2.07	MD FARHAN RAJA	354727D	2.33	REETHAM KUNDU	163763D	1.20
34	SHAHEED BANDWA	426226C	1.86	MD ARSHAD	304492D	9.33	RIYAZ AHAMED	739908D	1.95
35	SURYA S	927255C	1.50	MANIGANDA PRABHU	176030D	2.72	ROUNAK DAS	111085D	1.61
36	ANANDA GIRI	436775D	3.06	MONU SAHA	196809D	3.25	SADH K	723102D	1.81
37	BHABATOSH	390506D	3.13	MALIKUNZA'S BABY	552290B	4.10	SAISARAN	430882D	0.95
38	DEVA	453779D	2.86	PRIYANSHU RAHA	262311D	4.08	SENTHIL KUMAR	326936C	1.38
39	ESTIAQUE.A	458458D	0.87	PUSKAR SAHA	74395D	3.63	SHAIKH RIZWAN AHAMED	439122D	1.82
40	GAUTHAM	940577C	1.44	RITAM GHOSH	268410D	1.97	SHANKARI'S BABY	994140C	1.42
41	JHUMA BAGACHI'S BABY	239030D	1.63	RAJDEEP NATH	265281D	10.42	SHARMA S R	073749D	2.08
42	KUMARAN	406579D	1.00	SURYA PRATAP	177758D	6.12	SOUMYAJYOTI MAITY	437002D	1.33
43	MAYANK SHARMA	384236D	2.99	YRAVA VINEET	195361D	3.04	SOURAV DATTA	673064D	1.26
44	MOHD SAINAN	652058B	5.30	SAIKAT PAUL	438923D	4.00	SUBHRAPRAKASH ROUT	190432D	0.54
45	NILESH KUMAR OJHA	564805D	1.02	SAFWAN AHAMED	452157D	13.91	TAMBIR	694097D	0.94
46	PRITAM C	555344D	1.34	PRIANTU SAHA	462339D	8.23	TARUN S	638356D	1.40
47	RANJITH KUMAR	171030C	4.66	FATHIMAS BABY	501718D	11.36	USHA K'S BABY	720344C	1.05
48	SABITRIDEVI'S BABY	3970686C	3.89	ARNAB BAPLI	511503D	3.31	VIJAY T	674408D	1.00
49	SAMRAT GHOSH	481149D	4.93	VIMALAS BABY	850963C	5.62	VIJENDRA KUMAR	355576D	0.80
50	SAMU	598707D	5.23	ANIK BISWAS	436394D	3.84			
51	SANTHANA KUMAR	386855D	5.08	MANOSH DAS	585766D	8.35			
52	SHAHIL BURNWAL	426226C	2.76	SOUMYADEEP MANDAL	586906D	4.02			
53	SHARIERAAHAMED	491208D	1.22	JAYAS BABY	400541D	3.34			
54	SHUBHAM KUMAR	517658D	2.14	DIPAK KUMAR	410749D	5.71			
55	SUDIP KUMAR	499755D	3.10	AMERENDER PRATAP	425046D	3.00			
56	SUJAL GUPTA	440086D	3.05	SAYANDIP KANDAR	431737D	6.02			
57	SUSHANT SHURAV	444227D	2.82	RAHUL DAS	431761D	1.58			
58	TANMOY SAHA	396904D	2.82	PRIYAM J	335970D	2.34			
59	USHA'S BABY	955364C	5.66	JAYANTIS BABY	480013D	3.10			
60	VIJAY SARKAR RAMUL	512448D	1.64	SHIRINS BABY	498327D	8.58			
61	VIJAYALAKSHMI'S BABY	029535D	2.79	SHANU DATTA	503480D	4.89			
62	ADRITA DEB	743963C	3.92	DEBADRITO BHADURI	507299D	2.08			
63	DEPARPAN MUKHERJEE	642833D	2.40	EVA BAREHS BABY	556218D	8.37			
64	DINESH KUMAR YADAV	653744D	2.16	JIT SAHA	586810D	4.42			
65	JAMES	433405C	3.23	GOKUL S	390786D	2.71			
66	LALPEKHLUA	639592D	2.17	SULAIMAL NATCHI	646220D	4.62			
67	PRIYANSHU	683355D	3.67	SABARI VASAN	670673D	5.33			
68	RAGHAVI NATHAN	743295D	4.26	SUDIPTA MANDAL	703710D	5.98			
69	SABARINATHAN T	696075D	1.85	SUMAN CHAKRABORTY	576713D	2.07			
70	SHIVSAGAR SHAH	720259D	4.71	JUBIER AHMED	730893D	4.27			
71	TORIQUL ISLAM	683878D	2.44	BITTU RAY	740525D	3.14			
72	DEEPA'S BABY	499241C	3.92	CHAND ANSARI	781071D	4.02			
73	ARNAB HOTA	819651D	4.08	ARGHA DAS	791292D	10.39			
74				DIVYAS BABY	804789D	5.21			
75				KIRANDEVIS BABY	804221D	7.50			

NEUROGENIC BLADDER				PUV		
SR.NO	NAME	HOSP NO	RATIO	NAME	HOSP NO	RATIO
1	GEETHA'S BABY	589608C	0.91	DEBASHISH SHAW	129817D	5.38
2	LATHA'S BABY	244473D	1.23	DISANT PRADHAN	003841D	6.81
3	AMIT MATHEW THOMAS	242207D	7.50	DHANUSH	806392D	3.16
4	KAVIYARASU	369214D	7.18	AMAN VERMA	169767D	4.92
5	DEBU HALDAR	199189D	4.86	AYAN KONAR	790466C	9.22
6	MUNNA KUMAR	321693D	2.11	MANOMAY CHAKRABORTY	509241C	2.59
7	DHANRAJ SARVESH	083763D	2.23	SABARNA KARMAKAR	798701D	1.77
8	MD.SHAHEED ALAM	338592D	2.54	RAMANA	915292C	7.07
9	SATHISH M	109534C	1.86	PARUEZ BADSHA	087170D	7.23
10	SUMATHIS BABY	405224C	1.03	PRADEEP	961355C	7.89
11	TANMAY SAHU	315567D	1.11	NITYA BERA	104512D	4.19
12	YUVRAJ	202545D	8.44	NITHISH KUMAR	078495D	13.53
13	VELMURUGAN	833388B	6.46	YASWANTH	033455D	6.08
14	RANGARAJ	267157D	2.39	VINAYAGAM	630448C	1.06
15	PRABHAVATHI'S BABY	235725C	2.08	USHAS BABY	955364C	4.29
16	RAJU	986509C	2.78	SHRIKANT JANA	989392C	6.01
17	JAMSHEDUDDIN	019336D	2.13	SREEJAN KESARI	168445D	1.23
18	RAJKUMAR	815795C	0.80	SUMAN SUTRADHAR	156299D	3.08
19	AYUSHKUMAR VARMA	090466D	1.20	SHAMEERA BANUS BABY	073037D	2.56
20	MALAY BERA	099149D	1.18	SUBRAJIT DEB	077900D	6.40
21	AKASH SARKAR	822076C	4.41	ASHOK RANJAN S	885484C	3.05
22	GEETHA'S BABY	034947C	1.01	ANBU D	238246D	3.94
23	MUKESHKUMAR	024609D	2.12	ANJALI DEVIS BABY	885965C	4.56
24	LIYAKAT ALI	962196C	3.00	BABU	245960D	5.28
25	KONMAN	896802C	1.97	BISHAL GUPTA	162851D	3.11
26	KRISHNA SUMANTH	015891D	1.51	CHARAN K	562897C	3.81
27	VINAYAK	121664D	1.61	DEVIS BABY	233409D	5.49
28	VINEET VICTOR	036123D	3.23	IYANNER	308175D	12.26
29	SUDIP DAS	765698C	1.71	INZAMAMUL HAQUE	186789D	2.88
30	VIGNESH	522749B	5.56	JASIMUDDIN AHAMED	221705D	1.97
31	SELVI'S BABY	838989C	2.82	LOGNO SAHA	234192D	4.19
32	SWAGATAM DAS MANDAL	965407C	3.95	MOHITH NAYAK	368276D	7.12
33	TARNI SEN MANDAL	158360D	2.07	MD FARHAN RAJA	354727D	2.33
34	SHAHEED BANDWA	426226C	1.86	MD ARSHAD	304492D	9.33
35	SURYA S	927255C	1.50	MANIGANDA PRABHU	176030D	2.72
36	ANANDA GIRI	436775D	3.06	MONU SAHA	196809D	3.25
37	BHABATOSH	390506D	3.13	MALIKUNZA'S BABY	552290B	4.10
38	DEVA	453779D	2.86	PRIYANSHU RAHA	262311D	4.08
39	ESTIAQUE.A	458458D	0.87	PUSKAR SAHA	74395D	3.63
40	GAUTHAM	940577C	1.44	RITAM GHOSH	268410D	1.97
41	JHUMA BAGACHI'S BABY	239030D	1.63	RAJDEEP NATH	265281D	10.42
42	KUMARAN	406579D	1.00	SURYA PRATAP	177758D	6.12
43	MAYANK SHARMA	384236D	2.99	YRAVA VINEET	195361D	3.04
44	MOHD SAINAN	652058B	5.30	SAIKAT PAUL	438923D	4.00
45	NILESH KUMAR OJHA	564805D	1.02	SAFWAN AHAMED	452157D	13.91
46	PRITAM C	555344D	1.34	PRIANTU SAHA	462339D	8.23
47	RANJITH KUMAR	171030C	4.66	FATHIMAS BABY	501718D	11.36
48	SABITRIDEVI'S BABY	3970686C	3.89	ARNAB BAPLI	511503D	3.31
49	SAMRAT GHOSH	481149D	4.93	VIMALAS BABY	850963C	5.62
50	SAMU	598707D	5.23	ANIK BISWAS	436394D	3.84

51	SANTHANA KUMAR	386855D	5.08	MANOSH DAS	585766D	8.35
52	SHAHIL BURNWAL	426226C	2.76	SOUMYADEEP MANDAL	586906D	4.02
53	SHARIERAAHAMED	491208D	1.22	JAYAS BABY	400541D	3.34
54	SHUBHAM KUMAR	517658D	2.14	DIPAK KUMAR	410749D	5.71
55	SUDIP KUMAR	499755D	3.10	AMERENDER PRATAP	425046D	3.00
56	SUJAL GUPTA	440086D	3.05	SAYANDIP KANDAR	431737D	6.02
57	SUSHANT SHURAV	444227D	2.82	RAHUL DAS	431761D	1.58
58	TANMOY SAHA	396904D	2.82	PRIYAM J	335970D	2.34
59	USHA'S BABY	955364C	5.66	JAYANTIS BABY	480013D	3.10
60	VIJAY SARKAR RAMUL	512448D	1.64	SHIRINS BABY	498327D	8.58
61	VIJAYALAKSHMI'S BABY	029535D	2.79	SHANU DATTA	503480D	4.89
62	ADRITA DEB	743963C	3.92	DEBADRITO BHADURI	507299D	2.08
63	DEPARPAN MUKHERJEE	642833D	2.40	EVA BAREHS BABY	556218D	8.37
64	DINESH KUMAR YADAV	653744D	2.16	JIT SAHA	586810D	4.42
65	JAMES	433405C	3.23	GOKUL S	390786D	2.71
66	LALPEKHLUA	639592D	2.17	SULAIMAL NATCHI	646220D	4.62
67	PRIYANSHU	683355D	3.67	SABARI VASAN	670673D	5.33
68	RAGHAVI NATHAN	743295D	4.26	SUDIPTA MANDAL	703710D	5.98
69	SABARINATHAN T	696075D	1.85	SUMAN CHAKRABORTY	576713D	2.07
70	SHIVSAGAR SHAH	720259D	4.71	JUBIER AHMED	730893D	4.27
71	TORIQUL ISLAM	683878D	2.44	BITTU RAY	740525D	3.14
72	DEEPA'S BABY	499241C	3.92	CHAND ANSARI	781071D	4.02
73	ARNAB HOTA	819651D	4.08	ARGHA DAS	791292D	10.39
74				DIVYAS BABY	804789D	5.21
75				KIRANDEVIS BABY	804221D	7.50

CONTROL PATIENT (VUR)		
NAME	**HOSP NO**	**RATIO**
ADITYA KUMAR	217415D	1.06
AJAY PAUL	733425C	1.63
AMAN RAJ	782802D	1.02
ARIB NOOR FAROOQI	287000D	0.85
ARPIT RAJ	458298D	2.47
AYUSH KEDIA	386337D	1.18
BIKRAM RIT	331021D	1.76
BILOMBO BASAK	445039D	1.00
BIVEK KUMAR	704128D	1.30
CHITRAS BABY	629131D	1.08
DEEPAK M	210904D	1.03
DHEMAN ROY	254057D	0.98
DHINESH KUMAR	756190C	0.98
FERODES BEGAM'S BABY	859140C	0.92
GAURAV MASTER	594328D	1.17
HEENAKAR THAKUR	490448D	1.15
IBAN KITLANG	298651D	1.13
JASWANT S	472637D	1.17
KUTU	664852C	0.94
KRISHNENDU PAL	393686D	1.33
LACKI	116793D	1.03
MEERAJ MONDAL	535880D	1.16
MOHD AMASH	983190C	3.25
MOHD JASEEM	181991D	1.00
MOHD SHAHEEN	475612D	0.74
MRIGANKA GUHA	306039C	1.05
NANCY'S BABY	144633D	1.52
PRASANTH	903316C	1.77
PRETIVIRAJ	661842C	1.04
PUNITHA'S BABY	194272D	1.41
RAJDEEP PRAMANIK	709571D	0.89
RAJDITYA GHORAI	299781D	1.58
REETHAM KUNDU	163763D	1.20
RIYAZ AHAMED	739908D	1.95
ROUNAK DAS	111085D	1.61
SADH K	723102D	1.81
SAISARAN	430882D	0.95
SENTHIL KUMAR	326936C	1.38
SHAIKH RIZWAN AHAMED	439122D	1.82
SHANKARI'S BABY	994140C	1.42
SHARMA S R	073749D	2.08
SOUMYAJYOTI MAITY	437002D	1.33
SOURAV DATTA	673064D	1.26
SUBHRAPRAKASH ROUT	190432D	0.54
TAMBIR	694097D	0.94
TARUN S	638356D	1.40
USHA K'S BABY	720344C	1.05
VIJAY T	674408D	1.00
VIJENDRA KUMAR	355576D	0.80

SR.NO	HOSP NO	NAME	AGE	CAUSE OF NB	U.R	HN
1	589608C	GEETHA'S BABY	4	1	0.909091	2
2	244473D	LATHA'S BABY	1	1	1.234615	2
3	242207D	AMIT MATHEW THOMAS	3	2	7.5	2
4	369214D	KAVIYARASU	13	1	7.183099	1
5	199189D	DEBU HALDAR	0.5	1	4.860465	2
6	321693D	MUNNA KUMAR	12	1	2.105263	1
7	083763D	DHANRAJ SARVESH	3	3	2.232143	2
8	338592D	MD.SHAHEED ALAM	6	1	2.538462	1
9	109534C	SATHISH M	6	1	1.861111	1
10	405224C	SUMATHIS BABY	3	1	1.025641	2
11	315567D	TANMAY SAHU	14	2	1.105528	1
12	202545D	YUVRAJ	10	1	8.444444	1
13	833388B	VELMURUGAN	8	1	6.463158	2
14	267157D	RANGARAJ	10	1	2.391304	2
15	235725C	PRABHAVATHI'S BABY	6	1	2.082927	2
16	986509C	RAJU	5	1	2.78022	1
17	019336D	JAMSHEDUDDIN	7	3	2.125984	1
18	815795C	RAJKUMAR	1	1	0.8	2
19	090466D	AYUSHKUMAR VARMA	2	1	1.20202	2
20	099149D	MALAY BERA	1	1	1.18239	2
21	822076C	AKASH SARKAR	2	1	4.409091	1
22	034947C	GEETHA'S BABY	5	1	1.006623	2
23	024609D	MUKESHKUMAR	8	1	2.120482	2
24	962196C	LIYAKAT ALI	8	1	3	2
25	896802C	KONMAN	2	1	1.966292	2
26	015891D	KRISHNA SUMANTH	15	1	1.51375	2
27	121664D	VINAYAK	12	1	1.611289	1
28	036123D	VINEET VICTOR	6	1	3.227513	1
29	765698C	SUDIP DAS	2.5	1	1.706093	2
30	522749B	VIGNESH	11	1	5.560811	2
31	838989C	SELVI'S BABY	1	4	2.818182	1
32	965407C	SWAGATAM DAS MANDAL	2	1	3.945	2
33	158360D	TARNI SEN MANDAL	12	1	2.07029	2
34	426226C	SHAHEED BANDWA	3.5	1	1.864489	2
35	927255C	SURYA S	5	1	1.497485	2
36	436775D	ANANDA GIRI	8	1	3.055556	1
37	390506D	BHABATOSH	13	3	3.132552	1
38	453779D	DEVA	9	1	2.857143	1
39	458458D	ESTIAQUE.A	8	1	0.866667	2
40	949577C	GAUTHAM	4	4	1.441176	2
41	239030D	JHUMA BAGACHI'S BABY	1	1	1.628788	1
42	406579D	KUMARAN	2	3	1	2
43	384236D	MAYANK SHARMA	3	1	2.988372	2
44	652058B	MOHD SAINAN	6	1	5.302419	1
45	564805D	NILESH KUMAR OJHA	2	2	1.022222	2
46	555344D	PRITAM C	8	1	1.337079	2
47	171030C	RANJITH KUMAR	8	1	4.66055	2
48	3970686C	SABITRIDEVI'S BABY	5	1	3.891176	2
49	481149D	SAMRAT GHOSH	2	1	4.929293	2
50	598707D	SAMU	14	1	5.226415	1
51	386855D	SANTHANA KUMAR	9	1	5.075	2

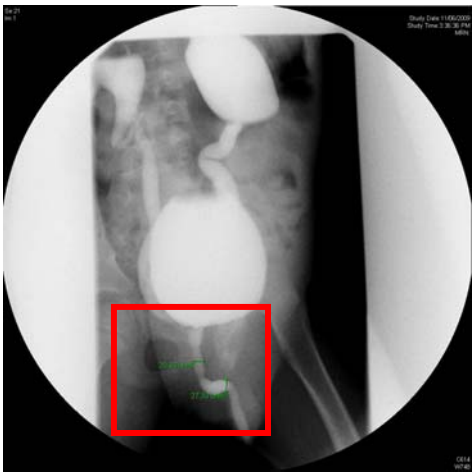
52	426226C	SHAHIL BURNWAL	5	1	2.757396	2
53	491208D	SHARIERAAHAMED	8	1	1.223404	2
54	517658D	SHUBHAM KUMAR	14	1	2.137931	2
55	499755D	SUDIP KUMAR	4	2	3.10101	2
56	440086D	SUJAL GUPTA	1	2	3.053846	2
57	444227D	SUSHANT SHURAV	5	1	2.817647	2
58	396904D	TANMOY SAHA	7	5	2.824818	2
59	955364C	USHA'S BABY	0.5	1	5.658824	1
60	512448D	VIJAY SARKAR RAMUL	4	1	1.64	2
61	029535D	VIJAYALAKSHMI'S BABY	2	1	2.7875	2
62	743963C	ADRITA DEB	8	3	3.919753	2
63	642833D	DEPARPAN MUKHERJEE	2	3	2.398333	2
64	653744D	DINESH KUMAR YADAV	10	1	2.163636	2
65	433405C	JAMES	6	1	3.234043	2
66	639592D	LALPEKHLUA	2.5	1	2.171429	2
67	683355D	PRIYANSHU	7	1	3.666667	2
68	743295D	RAGHAVI NATHAN	0.8	4	4.26	2
69	696075D	SABARINATHAN T	12	1	1.852174	2
70	720259D	SHIVSAGAR SHAH	14	5	4.707071	1
71	683878D	TORIQUL ISLAM	8	3	2.437403	1
72	499241C	DEEPA'S BABY	5	1	3.918083	2
73	819651D	ARNAB HOTA	1	1	4.081244	2

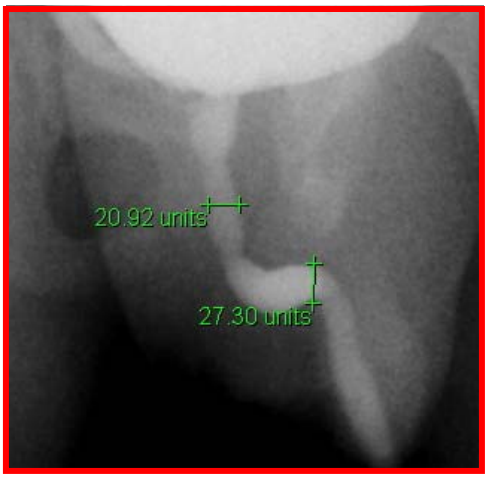
BLD TRAB	REFLUX	PU DILN	CREAT	PVR	TREATMENT	CMG
1	2	2	0.5	1	1	1
2	2	2	0.4	2	1	
1	2	2	0.5	1	2	
1	2	1	0.7	1	2	
2	1	1	0.4	1	1	
1	1	2	2.4	1	1	
2	2	2	0.5	1	1	
1	1	2	0.8	1	5	
1	2	1	0.6	1	5	
2	2	2	0.4	1	1	
1	2	2	0.6	1	2	
1	2	1	0.8	1	1	
1	2	1	0.6	1	1	
1	2	1	0.5	2	2	
2	2	1	0.6	2	6	
1	1	2	0.6	2	6	
1	2	2	0.5	2	1	
2	2	2	0.4	2	1	1
2	2	2	0.4	1	3	
2	2	2	0.4	1	2	
1	1	1	0.7	2	6	
2	2	2	0.5	2	4	2
1	2	1	0.7	1	1	
1	2	1	0.5	1	2	
2	2	2	0.4	2	1	
2	2	2	0.6	2	1	2
1	1	2	0.4	1	1	
1	2	1	0.5	2	2	2
2	2	2	0.4	1	2	
2	2	1	0.6	2	6	
1	1	1	0.5	2	1	
1	2	1	0.4	1	2	2
2	2	2	0.5	1	1	
2	2	1	0.4	1	1	
2	2	2	0.5	2	2	
1	1	+	0.6	2	6	
1	1	1	1.6	2	6	2
1	2	1	1.4	2	1	2
1	1	2	0.6	2	2	
1	1	2	0.4	1	5	2
2	1	1	0.4	2	5	
1	2	2	0.4	2	1	
2	2	1	0.5	2	2	
1	2	1	0.5	1	2	
1	2	2	0.4	1	1	
2	2	2	0.7	1	1	1
1	2	1	0.6	1	1	2
1	2	1	0.6	1	2	2
1	2	1	0.4	1	2	
1	2	1	0.6	1	2	2
1	2	1	0.5	2	2	

2	2	2	0.4	1	2	
1	2	2	0.6	1	2	
1	2	1	0.6	1	4	
1	2	1	0.6	1	4	1
2	2	1	0.5	2	1	
2	1	2	0.5	1	2	
2	2	1	0.6	1	4	
2	2	+	0.5	2	2	
2	1	2	0.4	1	2	
1	1	1	0.5	1	1	1
1	2	1	0.5	2	2	2
1	2	2	0.5	2	4	2
1	1	1	0.6	2	3	
1	2	1	0.5	2	1	2
1	1	2	0.5	2	2	2
1	2	1	0.6	1	1	2
2	2	1	0.5	1	1	
1	1	2	0.6	2	1	
1	2	1	1	1	2	2
2	1	2	0.6	2	2	
1	1	2	0.7	2	1	
2	2	2	0.4	2	3	2

P.U	A.U
21	23.1
32.1	26
60	8
51	7.1
41.8	8.6
24	11.4
25	11.2
33	13
6.7	**3.6**
12	11.7
22	19.9
30.4	3.6
61.4	9.5
33	13.8
42.7	20.5
25.3	9.1
27	12.7
22	27.5
11.9	9.9
18.8	15.9
67.9	15.4
15.2	15.1
52.8	24.9
24	8
17.5	8.9
12.11	8
30.26	18.78
61	18.9
47.6	27.9
82.3	14.8
27.9	9.9
7.89	**2**
28.57	13.8
53.66	28.78
26.79	17.89
55	18
34.74	11.09
40	14
6.5	7.5
9.8	6.8
21.5	13.2
25.3	25.3
25.7	8.6
52.6	9.92
10.12	9.9
11.9	8.9
50.8	10.9
39.69	10.2
48.8	9.9
27.7	5.3
40.6	8

46.6	16.9
11.5	9.4
24.8	11.6
30.7	9.9
39.7	13
19.16	6.8
38.7	13.7
48.1	8.5
16.4	10
22.3	8
31.75	8.1
28.78	12
35.7	16.5
45.6	14.1
22.8	10.5
25.3	6.9
42.6	10
21.3	11.5
46.6	9.9
15.77	6.47
25.35	6.47
40.69	9.97





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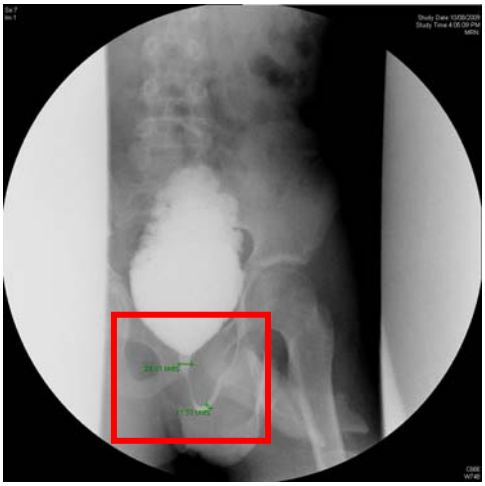

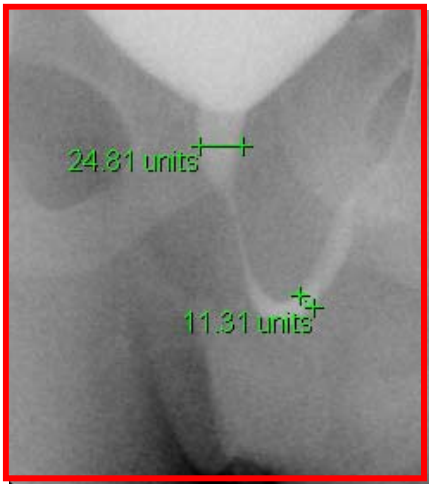

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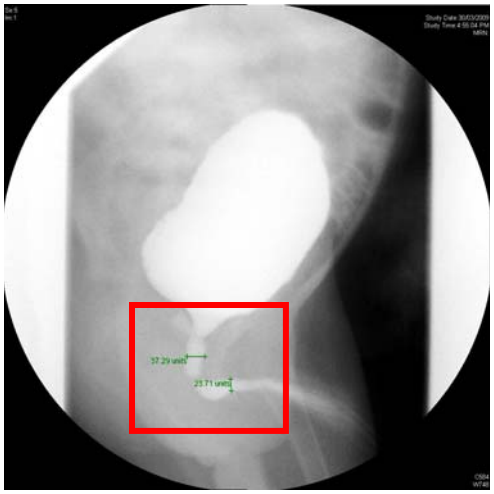
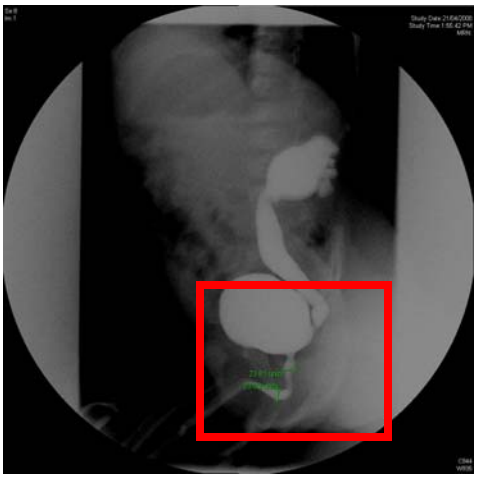
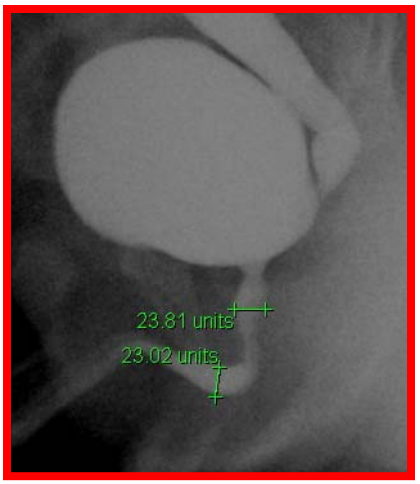


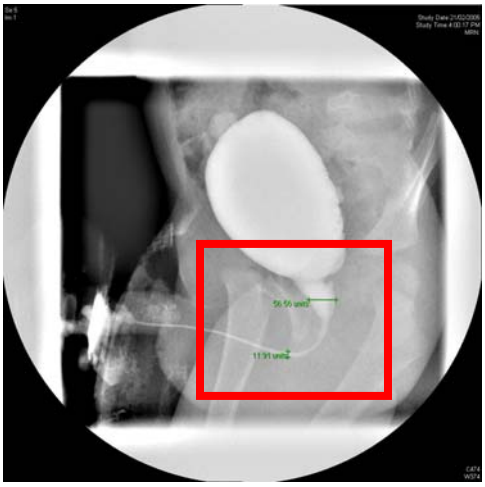
PUV

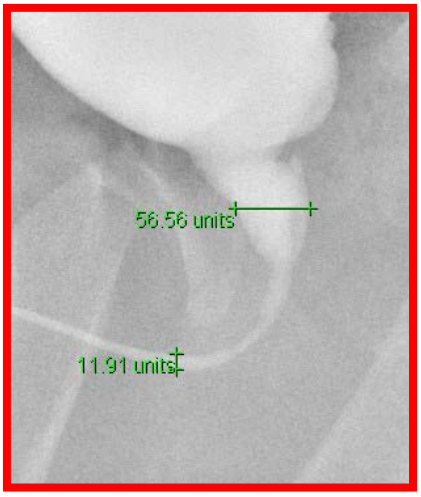
LOWER LIMIT OF URETHRAL RATIO IN EACH GROUP.





CONTROL





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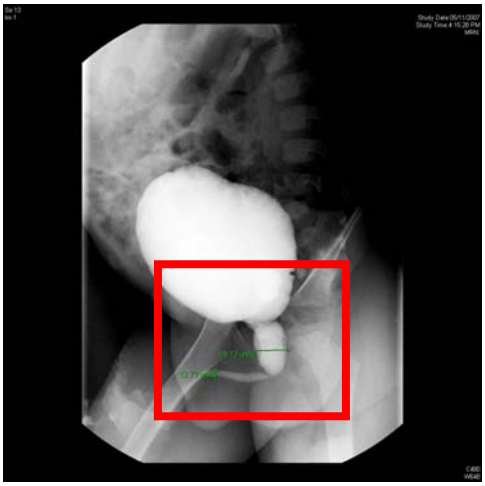
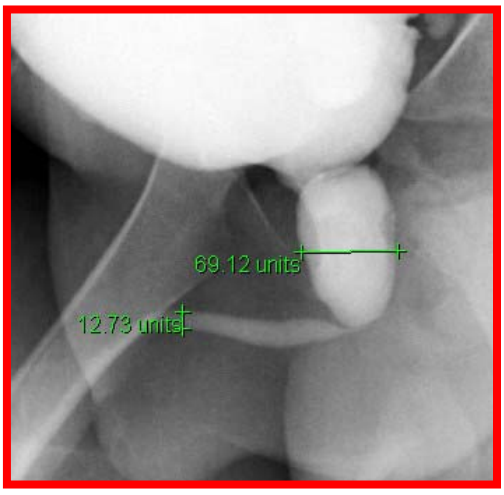
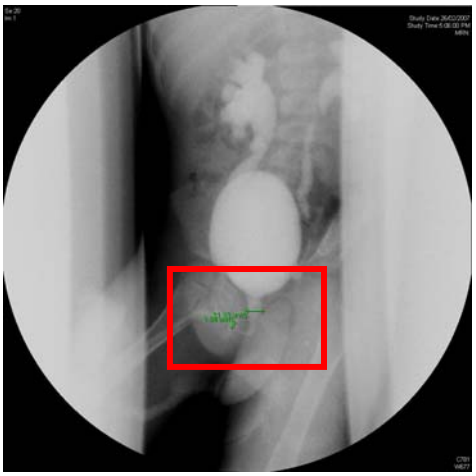
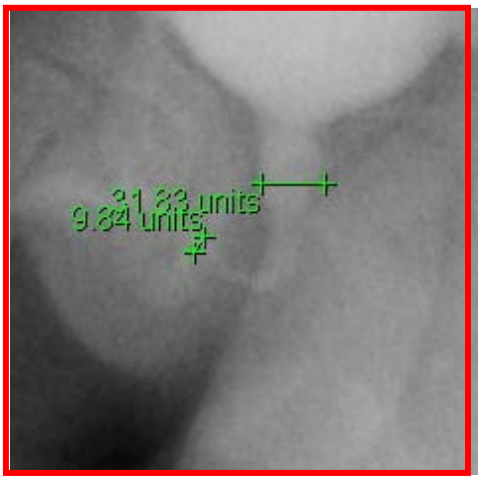




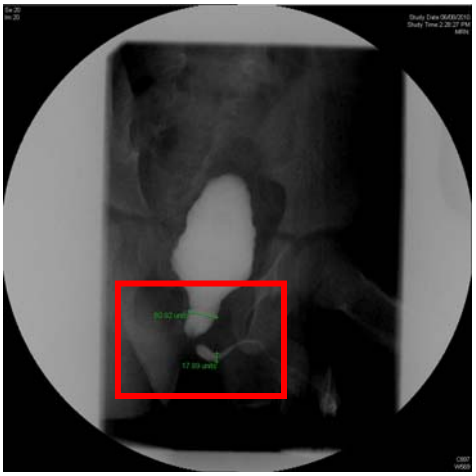
PUV

MEAN URETHRAL RATIO IN EACH GROUP





CONTROL



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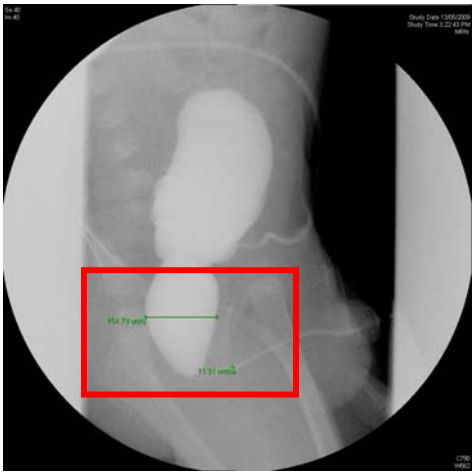
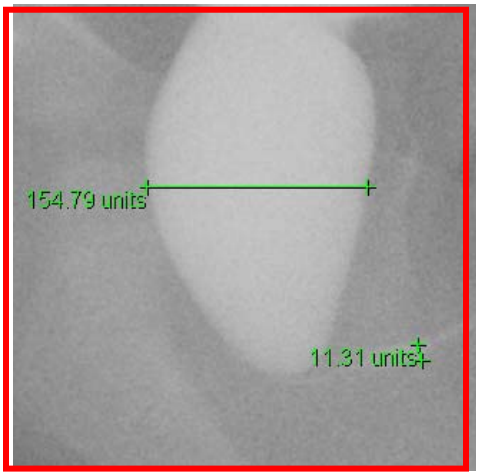
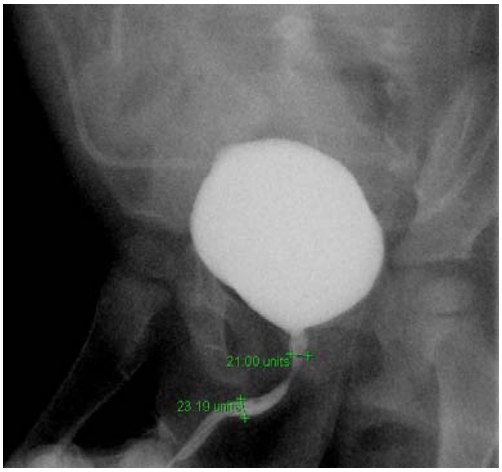




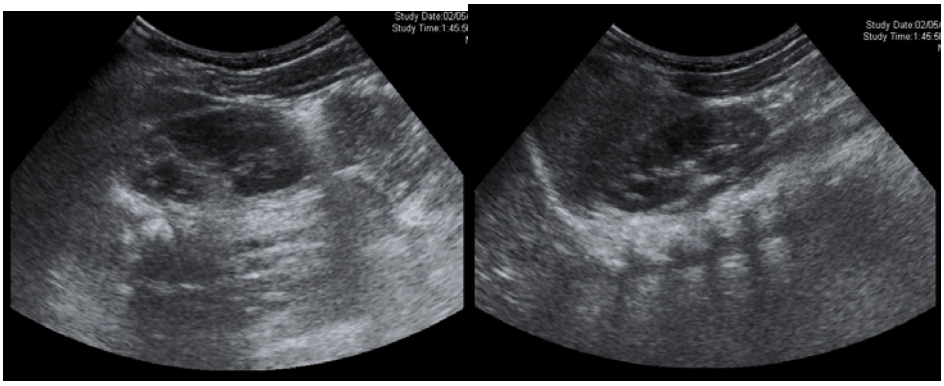
PUV

UPPER LIMIT OF URETHRAL RATIO IN EACH GROUP.

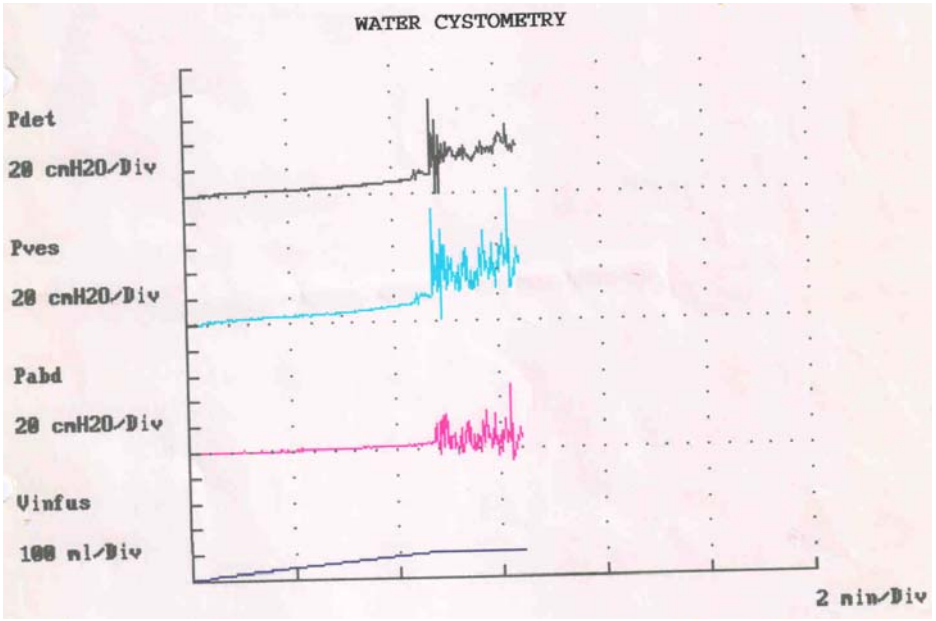
CORRELATION OF URETHRAL RATIO WITH USG AND CMG



MCU: Nondilated posterior urethra

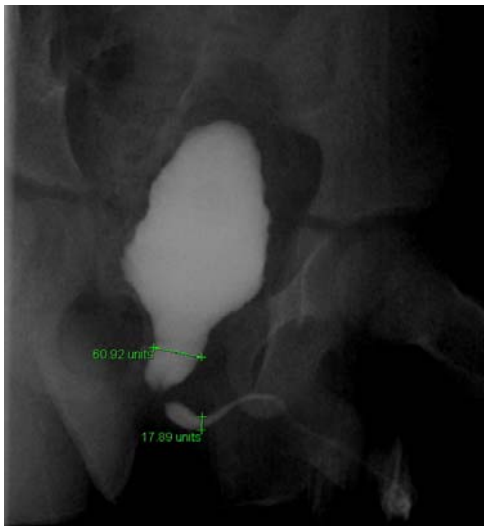


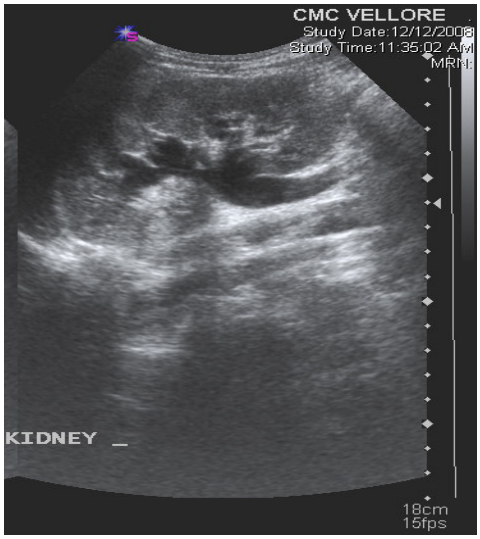
USG : No Upper Tract Changes



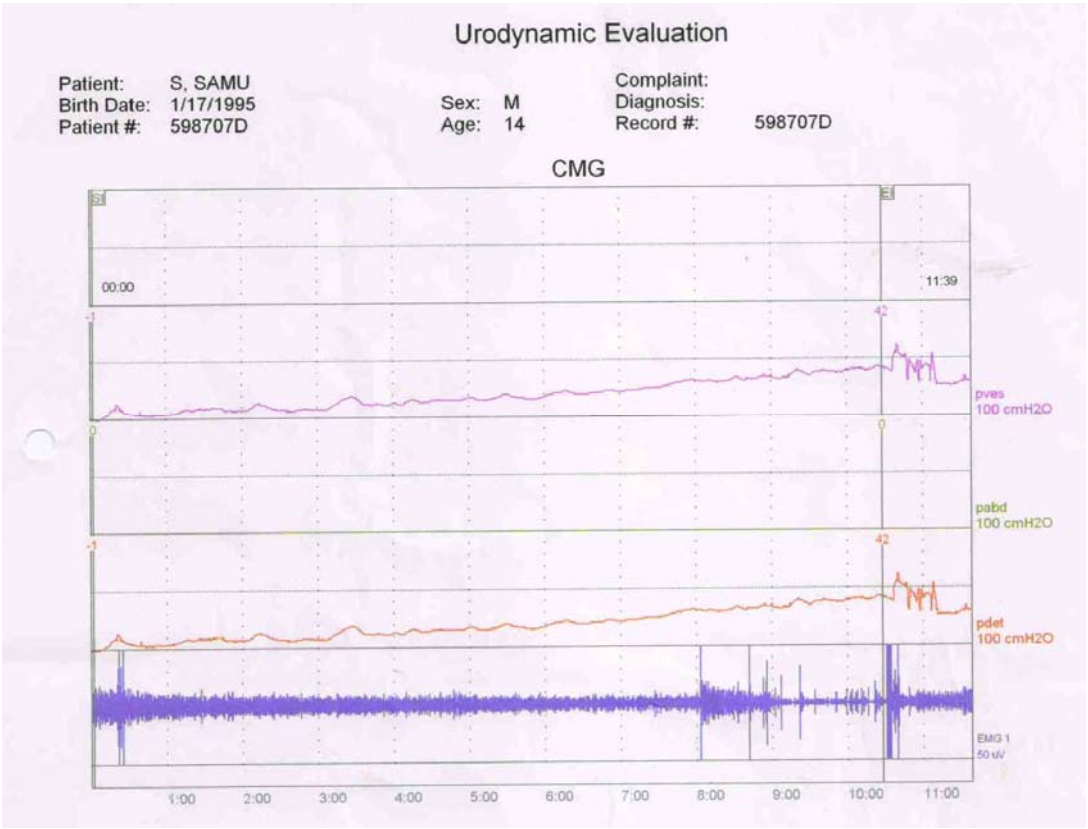
CMG: Good compliant Bladder

CORRELATION OF URETHRAL RATIO WITH USG AND CMG :





MCU: Dilated posterior urethra **USG:Hydroureteronephrosis**



CMG : Poor compliant ,high pressure bladder with DESD